Vol. I OCTOBER, 1917 No. 8

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THE AMERICAN REVIEW OF TUBERCULOSIS

JOURNAL OF

THE NATIONAL ASSOCIATION FOR THE STUDY AND PREVENTION OF TUBERCULOSIS

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PUBLISHED MONTHLY

AT 2419-21 GREENMOUNT AVE., BALTIMORE, MD.

BY THE NATIONAL ASSOCIATION FOR THE STUDY AND PREVENTION OF TUBERCULOSIS

Entered as second class matter March 9, 1917, at the post office at Baltimore, Maryland, under the Act of August 24, 1912

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THE AMERICAN REVIEW OF TUBERCULOSIS is issued monthly about the 20th of the month. Each volume consists of approximately 700 pages. The volumes begin with the March number.

The subscription price of the REVIEW to members of The National Association for the Study and Prevention of Tuberculosis is $2.00 per volume, and to non-members of the National Association, $3.00 per volume. Twenty-five cents extra should be added for Canadian and fifty cents extra for foreign postage. Subscriptions may be sent to 2419 Greenmount Avenue, Baltimore, Md., or to The National Association for the Study and Prevention of Tuberculosis, 105 East 22d Street, New York City. Checks should be made payable to William H. Baldwin, Treasurer.

Manuscripts should be sent to the office of the Managing Editor, Dr. Allen K. Krause, Johns Hopkins Hospital, Baltimore, Md. Manuscripts should be typewritten on one side of the page only. The publishers will not be responsible for manuscripts lost in transit. In order to save expense for authors' corrections in proof, the manuscripts should be carefully revised by the authors before submission.

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THE RÔLE OF SURGERY IN THE TREATMENT OF INTESTINAL TUBERCULOSIS

EDWARD ARCHIBALD

From the Surgical Service of the Royal Victoria Hospital, Montreal

The most dreaded complication of pulmonary tuberculosis is tuberculous enteritis or colitis. I think it is not too much to say that when signs of tuberculous trouble in the intestinal tract begin to make their appearance, and even before they are sufficiently definite for accurate diagnosis, both physician and patient become profoundly depressed and are ready to throw up their hands in despair.

In the mind of any one interested in tuberculosis from the surgical side, the consideration of this aspect of the disease can not fail to call forth the query, can anything be done to save these poor people from the sure fate awaiting them, or if not that, to relieve in some measure their distressing abdominal symptoms? Theoretically, a short-circuiting with exclusion of the diseased part of the bowel to give it rest, seemed promising. My first patient, referred by Dr. E. R. Baldwin about six years ago, was much relieved of pain by an ileo-sigmoidostomy, though she died later of generalized tuberculosis. With this encouragement, and particularly with Dr. Lawrason Brown's moral support, other cases were operated on, with the result that during the last three or four years I have treated twenty-seven cases of tuberculosis of the intestinal tract, including the appendix, and now feel that the experience is sufficient to justify its publication. In addition I have found in the records of the Royal Victoria Hospital in Montreal, ten other cases operated on by Dr. Garrow, Dr. Armstrong, and Dr. Keenan, who have kindly allowed me the use of their records. I include also three cases of Dr. Robert C. Paterson's, of Saranac Lake, who has been kind enough to give me his notes. My own cases have been sent to me chiefly by Drs. Brown, Kinghorn and Baldwin of Saranac Lake, and Dr. Byers of Ste. Agathe, to all of whom my thanks are due.

Tuberculosis of the bowel is ordinarily divided into the hypertrophic and the ulcerative types; and the French add a third, which they call

the fibrous or sclerotic type meaning really an advanced stage of scarring, following the ulcerative type. From the surgical standpoint, to judge from the literature, it is chiefly the hypertrophic tuberculosis of the caecum, and the stenosing fibrous lesion of the small bowel leading to obstruction, which hitherto have brought the patients to operation. A considerable number of case reports have been published concerning the hypertrophic caecal disease, and in that form the results of resection have been quite good. A fair number also of cases of tuberculous stricture of the small bowel have been published with results decidedly less good. Finally, the subject of tuberculosis of the appendix has received a good deal of attention of late years, but ultimate results are little known.

The present series includes only one case of the hypertrophic type, and that involved the appendix alone; likewise, only one case of stricture causing partial obstruction. All the rest were of the ordinary ulcerative type. These cases, so far as I can judge from the literature accessible to me, have received hitherto almost no consideration from the surgeon.

On the pathological side, the frequent opportunity of examining the bowel *in vivo*, at a stage usually long before postmortem, has resulted in our reaching a few conclusions which may be, somewhat categorically, set down. In the majority of our cases the caecum was diffusely thickened and easy to palpate, whether the small bowel or the rest of the colon was involved or not. The caecal disease is the oldest lesion, and it probably spreads from here both up and down the bowel, as was indeed observed in two re-laparotomies six months after a first operation. This is important from the surgical standpoint, if we can succeed in diagnosing the cases earlier. The rest of the colon is rarely so diffusely involved as to result in palpable thickening; it bears isolated ulcers that are more or less numerous. The ascending colon, the second part of the transverse colon, and the sigmoid are most apt to be ulcerated; while the two flexures and the first part of the transverse colon and the descending colon are frequently free from disease. The rectum is rarely involved without the anus being similarly affected.

At operation, it is usually possible to recognize the limits of the disease in the bowel; a mucosal ulcer or a tubercle in the wall nearly always betrays its presence by a patch of reactive fibrino-plastic exudate on the serosa and a local thickening of the wall.

DIAGNOSIS AND CLINICAL SYMPTOMS

With regard to the diagnosis of tuberculous disease in the intestinal tract, what has particularly impressed me has been the apparent unwillingness of the general medical men to come to such a diagnosis. I believe this is partly psychical. They probably hate to face the possibility of it. And yet early diagnosis is very desirable, because undoubtedly surgery can in selected cases accomplish a great deal; if operation is long delayed for lack of diagnosis the patient's lungs are apt to grow progressively worse *pari passu* with his intestinal disease; and when operation comes to be done, there is the greater danger from the side of the lungs.

In the presence of suspicious symptoms, we have to inquire first, whether tuberculosis has attacked the bowel; and if so, secondly, what part of the bowel is involved.

First, has tuberculosis invaded the bowel tract? In all the cases of my analysis, it was possible to trace certain general symptoms, which might be reasonably considered, at the onset at least, as indicating some one of the conditions of dyspepsia or derangement of the motor functions of the tract so common in the sedentary life of the phthisical person, especially in those on whom overfeeding is imposed. Such general symptoms are briefly, constipation, flatulence, gas in the bowels, slight loss of appetite, a feeling of weight or discomfort in the stomach after eating. Some or all of these are constantly present in patients in whom tuberculous intestinal disease is beginning. Of themselves, they do not naturally give the diagnosis. But after these come special signs or symptoms. The chief early symptom which should arouse our suspicion is pain. Your ordinary dyspeptic may have epigastric pain suggestive of peptic ulcer in the stomach or the duodenum but he does not have pain below that region. When the patient complains of a pain felt in the mid or lower abdomen, coming on at irregular intervals during the day, but chiefly from the late forenoon or afternoon on, transient often, crampy or stabbing, suggesting gas pains, aggravated by food and relieved by fasting, felt only during a part of the day, but persisting from day to day, then one must be very suspicious of tuberculosis. When in addition, he complains of loss of appetite, of real distaste for food; when he has nausea at times; when he gives up one article of food after another; when he develops a slight fever which is not

attributable to his lung condition, and if this persists over three or four weeks, then one may be almost sure of the diagnosis.

It remains to determine what part of the bowel is affected and to what extent.

In order to determine this, if possible, I have divided the cases for analysis into seven classes. First, those in which the appendix alone is involved; second, those in which in addition the caecum with or without a part of the ascending colon is diseased; third, those with disease in the caecum and some part of the rest of the colon, the rectum remaining free; fourth, those in which a large part of the colon with the rectum is involved; fifth, those with disease of the small bowel; sixth, those with disease in both small and large bowel; and seventh, those in which suppuration of the mesenteric glands is the only lesion, the bowel being healthy.

Of the first class, those in which, so far as we can tell, the disease was confined to the appendix, I have had seven cases. The outstanding clinical feature about these is that they give symptoms comparable with those of a recurring subacute appendicitis of the ordinary type, except that the picture is much less well defined. A really acute attack like that of ordinary frank appendicitis was found in only one case, in which operation showed the presence of acute ordinary inflammation grafted upon a chronic tuberculous condition. All the others gave rather indefinite symptoms of dull pain with soreness in the right lower quadrant, coming on frequently in mild attacks, but leaving persistent tenderness.

The second class, in which the caecum was involved in addition, numbers eight cases. The history is no longer that of a plain appendicitis; it is usually characterized by indefinite crampy pains, at first generalized, later becoming gradually localized in the caecal region, coming on at intervals, leading ultimately to more or less constant localized soreness, the pain being usually in direct relation to the taking of food, usually some two or three hours after meals and nearly always with distressing flatulence. Constipation is the rule at first, but later when the caecum becomes considerably involved, diarrhoea is apt to begin and may alternate with constipation or become more or less constant, but rarely with more than two or three stools a day. Anorexia is an early and persistent symptom unless the caecal wall is very little involved. Nausea is frequent, but vomiting is rare unless a subacute appendicitis is superadded. Pain seems to be due chiefly to gas and is consequently apt to be transient, recurring and colicky, and to come on in the afternoon

and disappear in the late evening. On examination it is possible to make out with a little practice a definite thickening of the caecal wall, which is tender on deep pressure. This was found in practically all of this series with the exception of one in which the involvement of the caecum was very slight, and in this case there was tenderness without mass. The chief diagnostic points are the diarrhoea and the palpable thickening.

In the third class, in two cases, the caecum and beginning of the ascending colon were chiefly involved, but there were isolated ulcers in the transverse colon, and in one also in the sigmoid. These give practically the same symptoms as the preceding class. The addition of isolated ulcers in the lower colon seems to add nothing to the symptom-complex.

In the fourth class the chief signs depend on involvement of the rectum. Accordingly we have an onset with diarrhoea, with early appearance of pus and blood in the stools, and pain in the rectal region. The proctoscope gives the diagnosis immediately, but if the caecum is involved it is possible to make certain of this also by palpation.

In the fifth class, in which the small bowel alone is diseased, I have only one case of my own, but can add two others who were operated on by other surgeons. The case reports leave one in slight uncertainty as to the exact extent of the lesion. In these, constipation seems to be marked; in none of the three is diarrhoea mentioned, and in two stress is laid on the constipation. The diagnostic picture is that of transient, crampy gas pains, not severe, generalized, felt irregularly here and there, together with troublesome anorexia, flatulence, occasional nausea, and slight distention. All these symptoms are persistent and resist treatment, but are relieved by fasting. With a picture of this sort, and in the absence of tenderness or thickening in the caecal region, one must be very suspicious of disease confined to the small bowel.

In class six, in which both small and large bowel were affected, there were fifteen cases, of which thirteen are available for analysis. These all show the general symptoms of pain, gas, anorexia, flatulence, and often slight fever not due demonstrably to the lung condition. But in addition the most interesting thing worked out concerns the occurrence of constipation and diarrhoea. These cases can be divided into three groups.

First, those in which the small bowel was chiefly affected, while the caecum and colon were only slightly diseased.

Second, those in which the caecum, with or without the ascending colon, was chiefly diseased, while the small bowel showed only a few ulcers.

Third, those in which both small and large bowel were considerably affected, but in which one or the other predominated.

In general it may be said from this analysis, that with very few exceptions diarrhoea is more or less proportionate to the extent of involvement of the caecum and ascending colon, while constipation characterizes those in which the disease is confined to the small bowel. A slight involvement of the small bowel may be unaccompanied by constipation, but marked disease in either direction seems practically always to be accompanied by constipation or diarrhoea according to the case. Alternating constipation and diarrhoea nearly always indicates involvement of both small and large bowel.

When we add to this a consideration of the palpatory findings, we are able to establish a scheme of general diagnosis which I have condensed in the following way. Five classes may be established:

First, when the small bowel alone is involved, there is present constipation, often quite marked, and there is no thickening or mass, and in particular no tenderness to palpation localized in the caecal region.

Second, when the caecum with or without the ascending colon is involved, and the small bowel is free, there is present diarrhoea or alternating constipation and diarrhoea, and there is always to be felt a fairly defined thickening, sometimes even a mass, and there is tenderness to palpation in the caecal region.

Third, when both small bowel and caecum are involved there is a mixture of the above signs and symptoms.

Fourth, when the transverse and descending colons are diseased in addition, there are very rarely any signs that point to it; these are comparatively silent regions.

Fifth, when the appendix alone is tuberculous, it is not possible before operation to exclude the possibility of disease in the caecum; one can only say that there is disease in that region. Between simple appendicitis and tuberculous disease it is often difficult to make a diagnosis; but it may be said that, where definite thickening of the tissues in the appendix region is felt, without there being an acute inflammatory reaction, and with a history of chronic slight pain and of occasional subacute attacks with persisting soreness, then the condition is much more suspicious of tuberculosis of the appendix, and possibly also of the caecum, than of simple appendicitis.

In the seventh class I have two cases in which the bowel was not involved and the condition consisted of a large mass of breaking down tuberculous glands in the mesentery. In one the mass was as large as an orange, and was situated in the root of the mesentery overlying the superior mesenteric artery. In the other it was a little lower in the mesentery at the level of the umbilicus under the right rectus. The symptoms in both these cases differed strikingly from the bowel ones in that, while the patients suffered from general abdominal distress with flatulence, they both had pain definitely localized to the area of the mass, never crampy, but rather burning and throbbing. Tenderness to palpation was accurately localized to the region of the mass. One had high fever and chills, both lasting for a long time before and after operation.

Feeling that the barium meal ought to give information of value in these cases, I asked Dr. Pirie to investigate the matter, and I have some ten examinations to report, all of which confirm his statements. He writes as follows:

X-Ray Diagnosis (by Dr. Pirie, Roentgenologist to the Royal Victoria Hospital)

Dr. Archibald has asked me if I could write from memory a summary of my findings in tuberculous conditions of the intestines. I do so with pleasure, as it was the most interesting work I did for about a year before the war began. Writing as I do without notes from a base hospital in France, I well remember the disappointment we had as in several successive cases of tuberculous caecum I failed absolutely to help in the diagnosis. Some bad luck seemed to dog my steps, for whereas we had formerly been able to show by means of a barium meal the position, shape and size of the normal caecum, yet we failed, in these tuberculous cases, to catch the caecum at the proper time when it was filled by the meal. Finally, the cause of our failure struck us. We could not show the caecum filled by a barium meal because it never did fill when tuberculous ulceration existed. Setting out with this as a working hypothesis, we examined the cases at half-hour intervals from four to twelve hours after the barium meal. We found our theory correct in each case of tuberculous caecum, namely, the caecum never filled up with the meal. Each small squirt of barium that left the ileum was quickly passed on past the caecum and collected elsewhere in the large colon, either in the transverse or the descending part. So that our conclusion was, that in a tuberculous subject with symptoms suggesting tuberculous caecum, if the caecum did not fill from four to twelve hours after the barium meal when examined at intervals of about half an hour, then this want of filling confirmed the diagnosis. On

the other hand when it did fill as it does in a normal individual, it negatives the diagnosis of tuberculous caecum. We had this latter experience confirmed in one case where a clinical diagnosis of tuberculous caecum had been suggested; the X-ray showed a caecum filled by barium, and at operation a non-tuberculous caecum was found.

RESULTS OF OPERATION

Results of operation for tuberculosis of the alimentary tract can and ought to be viewed from two different standpoints; one is the extent of disease at the time of the operation; the other is the degree of lung involvement. Of course it may be said in general that the more extensive the disease in either aspect, the worse must be the results of operation. A third point of view concerns the results in so far as palliation of distressing symptoms as the result of partial or radical operations is involved.

With regard to the extent of intestinal tract involvement, I have taken the same seven classes as for the clinical analysis.

First, those in which only the appendix is tuberculous. Of these I have seven cases. As to the immediate results, it may be said that all of these were relieved of the symptoms which brought them to operation, that is, the pain or abdominal distress, nausea, or vomiting, and the flatulence disappeared. The results are, on the whole, not very far behind those which we are accustomed to get in ordinary appendicitis cases, and the patients declare themselves well satisfied.

With regard to the late results I have information in five only. Of the other two, one is a recent case. Of these five, one (R. W.) died some four years after operation of her pulmonary lesion, having developed no signs of progressing intestinal disease. The other four are well from four to eight years after operation. In view of these facts I think we must revise the generally held opinion that tuberculosis isolated to the appendix is extremely rare. It is hardly likely that these patients had tuberculosis elsewhere in the bowel.

Second, those in whom the disease involved the appendix, caecum, and possibly a part of the ascending colon. Of these I have eight cases of which five were treated by resection of a few inches of the terminal ileum together with the ascending colon, and the first part of the transverse colon. Of these five I have late results in all, from two to four years after operation. All are dead but one, who is living, well and working, three years after operation. He, however, had no pulmonary dis-

ease. One other, without pulmonary disease, got complete relief from diarrhoea, pain and nausea for seven months, and then died in a few days of acute intestinal obstruction. Two others were completely relieved of their distressing abdominal symptoms, enjoyed their food and lived for about one and a half years. Both died of advancing pulmonary tuberculosis. As to the fifth, he was relieved of his abdominal symptoms, but after discharge from the hospital was lost sight of. It is known that he died at his home eight months after operation with symptoms of a brain lesion.

The other three cases of this series of eight showed chiefly appendicular tuberculosis, the caecum being involved only for a very small area adjacent to the base of the appendix. In these cases only the appendix was removed. One (Wilcox) reports twenty months later that he is only partly relieved of his abdominal symptoms, but that his lungs give him the chief trouble. Two others (Strauss and Ayotte) died in the course of the next six months to a year, of advancing pulmonary disease. In one of these, it is known that diarrhoea persisted. As to the other, I have no information.

In the third class I have two cases in which the caecum, ascending colon, transverse colon and sigmoid were involved with scattered ulcers. In both of these a resection with short-circuiting was done. In the one (Trumbull) three inches of the ileum and all of the colon up to the left edge of the transverse colon were resected, and in addition an isolated ulcer in the sigmoid was locally excised, apparently eradicating the disease. Short-circuiting was done between the ileum and the sigmoid 4 inches below the situation of the ulcer mentioned. This man made an excellent recovery, was entirely relieved of most distressing pain, nausea and disgust for food, gained 35 pounds, and was able to go around with some freedom, living in comfort for fifteen months. His lungs, however, had originally been seriously diseased, there was scattered trouble throughout the entire right lung and through the upper lobe of the left, and he had had haemorrhages a year before operation. Fifteen months after operation he suddenly had a severe haemorrhage which was followed by ten others in two weeks. His lungs, which seemed previously to be quiescent, went bad, and he died within five weeks, without any return of bowel symptoms.

In the second case the appendix, caecum, and part of the ascending colon were extensively involved, with a few ulcers in the transverse colon. At a first operation the appendix alone was removed, as the

patient refused anything more extensive. Nine months later, when his pulmonary and general condition were both much worse, he consented to a resection of the ileum to the middle of the transverse colon. Unfortunately the anaesthetist (against my wish) had given him some ether throughout the operation. He had some pulmonary oedema immediately following, which went on inside of ten days to an acute tuberculous pneumonia of the lower lobe of his good side. He was relieved to a large extent of his abdominal symptoms, but never recovered from the pneumonia, and died within a few months of progressive pulmonary tuberculosis.

In a fourth class, two cases, both without pulmonary disease, a great part of the large bowel only was diseased. In one the rectum and sigmoid were involved and he was cured by a left sided artificial anus kept open for a year. In the other, the disease involved the caecum and the left half of the transverse colon together with all the descending colon, sigmoid and the rectum. Here the caecum was resected and an artificial anus established on the right side. This is still open after three and a half years and the patient is extremely well. In both these cases the process of healing so contracted the colon as to make it unfit for further use. In the first case an attempt was made to extirpate the lower descending colon, sigmoid and rectum, and to bring down the upper half of the descending colon to the anus. This man unfortunately died of operative shock. The other man's caecal artificial anus will have to be permanent, but he is very comfortable, and is about to marry.

In the fifth class (three cases) the abdomen was explored and the disease found to occupy the small bowel only, but very extensively. Such cases might well be considered a *noli ne tangere*, and this with justice. There are isolated reports in the literature of strictly localized tubercular disease in the small bowel, usually causing stricture and operated on for obstruction, and some of these have been cured by operation. I am not here considering this type of case. The three patients referred to all had tuberculous ulcers scattered over a considerable part of the intestine. In one the abdomen was closed and nothing was done; the later history is not available. In another an ileo-sigmoidostomy was done. This patient was completely and permanently relieved of her abdominal symptoms which had been fairly severe, but died within five months of progressive pulmonary disease. In the third patient I found the ulcers confined to the lower $4\frac{1}{2}$ feet, by measurement, of the

ileum. I short-circuited the ileum into the transverse colon, excluding $4\frac{3}{4}$ feet of the terminal ileum. He too, was made comfortable as far as his abdominal symptoms were concerned, but his lungs immediately became active and he died in two months of his pulmonary tuberculosis. Before the operation his right lung was completely fibrosed with a large cavity, and the left apex was involved, but the latter condition was understood to be stationary.

In the sixth class there were fifteen cases, divided as follows: In seven nothing was done save an appendectomy, leaving behind extensive disease, and all of these died in from a few months to a year and a half, from the double load of lung and bowel disease, but in five of them there was considerable relief from pain for several months. In four a palliative operation was done, short-circuiting the ileum to the sigmoid, in three with exclusion, and in one by a side to side anastomosis. All were greatly relieved of their distressing symptoms. In two the relief was permanent, one dying of acute obstruction five months after operation, the other living and well two years after operation. In the other two some abdominal distress recurred several months after operation, and both died of their pulmonary condition within six months.

A radical removal of the disease by resection was done in four. In one relief was permanent for eleven months at which time he apparently got ptomaine poisoning, and died with some diarrhoea and serious aggravation of his lung condition. The second, in whom I resected $5\frac{1}{2}$ feet of the ileum and the colon up to the transverse portion, at first got relief, which was followed by recurrence of his diarrhoea. His lungs after the first month began to show serious aggravation and he died in four months of his pulmonary lesion together with a serious diarrhoea due to the exclusion of so much ileum. The third got permanent and complete relief, and is alive and well six years later with a very early pulmonary lesion. The fourth died three days after operation, probably of adynamic ileus.

RELATION OF PULMONARY LESION TO SURGICAL RESULTS

Viewing the results from the standpoint of the extent and character of the pulmonary disease, one finds in general as was to be expected, that the best results are obtained where the lungs are only slightly affected or are free of disease, while the worst results are seen in the patients with advanced and progressive lesions.

There are eight cases without pulmonary disease. Of these, two had tuberculosis of the appendix only, which was removed. Both are alive and well two years and six years after operation. In three both caecum and appendix were affected, and a resection was done in all. Of these, two are alive and well two and six years later, but one has developed incipient trouble in one lung. The other one was perfectly well for seven months, and then died of an acute obstruction. In two others there was a large mass of suppurating tuberculous glands in the root of the mesentery. The mass was incised and drained in one, and the patient was alive and well three or four years later. In the other, the mass was excised together with 4 feet of the ileum, with a perfect result, but after six months of good health, he died suddenly, the cause being unknown. Finally there were two patients with extensive tuberculous colitis and proctitis. An artificial anus giving rest to the bowel gave a splendid result. One died a year after operation from the shock of an operation designed to excise the scarred and contracted bowel; the other is well and at work three and a half years after operation, but with the artificial anus still open. Thus of eight cases, five are alive and three have died, but all from causes unconnected with any extension of their disease.

Of those in which the lungs were only slightly affected, there were five cases. In three the appendix alone was diseased, and two are living and free of abdominal symptoms, but are still, after several years, being treated for their lungs while one died of unknown cause a year or two after operation. In another case the disease was extensive through the ileum, and the appendix was also involved. In this case an ileo-sigmoidostomy was done (Dr. Paterson's and Dr. Bazin's case) and the patient, after prolonged and complicated convalescence, is alive and well two years later with his lungs still in fair shape. This is a remarkable case, showing the possibility of healing of tuberculous bowel, if some help is afforded. The fifth case had extensive disease throughout the small bowel for which no palliative operation was done. The result is unknown but she probably died within a short time.

When we come to the class including those in which the lungs showed extensive or advanced disease which was chronic, stationary, or very slowly progressive, the results immediately get worse. Of these there are ten cases. Among these are five resections of the terminal ileum and the colon as far as the transverse portion, apparently eradicating the disease. These have all died, one of adynamic ileus three days after operation; a second of presumable ptomaine poisoning with progressive

lung disease fourteen months after operation, after having been in splendid health for a year; a third of advancing pulmonary disease probably caused by the resection of 5½ feet of the terminal ileum with consequent excessive loss of absorbing surface and chronic starvation; the fourth and fifth of progressive lung trouble, the intestinal symptoms not recurring. In three others the disease was too extensive in both small and large bowel for radical operation, the appendix being removed in two, and a short-circuiting with exclusion of 4½ feet of the terminal ileum in the other. This last patient died in three and a half months from aggravation of the pulmonary disease plus partial starvation from too extensive exclusion of the ileum. The others died of progressive generalized disease. Finally there are two cases in which the appendix only seemed to be involved: of these one died four years later from her lung disease, while the other is alive six years later, and is well, although a chronic case.

In the fourth class in which the pulmonary disease was not only advanced but active and progressive, there were fourteen cases of which only one is now living, and that is a recent case. Among these there were six palliative appendectomies, and four ileo-sigmoidostomies; all for conditions of disease affecting the bowel too extensive to permit of resection. Although many of these obtained great relief from their abdominal symptoms, they all died in from three to twelve months, usually of progressive disease in lungs and bowels, although one died of acute obstruction five months after operation, having had great relief till then. There were three resections of the ileum to the transverse colon, eradicating the disease, and although all three of these died, the results as far as the abdominal condition was concerned were very encouraging. All three had permanent relief from abdominal symptoms, but one died sixteen months later, and another in fourteen months, from their pulmonary disease and haemorrhages. The other died in a few months from aggravation of the lung trouble caused by the anaesthetic.

I would call attention to the remarkable effect which the removal of the appendix alone has often exercised in cases where the rest of the bowel may be extensively diseased. Frequently pain disappears and likewise nausea, flatulence and anorexia. This good result often lasts for several months. It is decidedly worth while, even where the disease cannot be eradicated, to take out the appendix as a measure of palliation. However, one must be prepared to expect that other symptoms,

particularly diarrhoea or constipation, will not be relieved by an appendectomy, but from the patient's point of view these troubles are very bearable as compared with the pain from which he has been freed.

MANAGEMENT OF OPERATION

I think it is now generally accepted that the safest anaesthetic for pulmonary cases is nitrous oxide gas with oxygen. I think I may say that I have never found it to irritate the lungs. In a series of thirty operations of my own, I used gas-oxygen with local novocain infiltration twenty-four times, in six of which it was necessary to add a little chloroform or a little ether; in three ether alone was used; in one, chloroform alone; and in two, local alone. With care and patience it is nearly always possible to secure excellent anaesthetic conditions for the very extensive operations which have frequently been performed.

I might add to this series a further list of over seventy operations on tuberculous subjects, most of them for appendicitis, in which gas-oxygen has been employed almost exclusively. In all these cases the effect of gas-oxygen on the lungs has been nil. It is true that in five of the present series there occurred a definite aggravation of the pulmonary disease following this anaesthetic, but in none of these could it be ascribed to the anaesthetic. In all it occurred some two or more weeks after operation, and in two it was certainly due to partial starvation as the result of an exclusion of $4\frac{1}{2}$ and $5\frac{1}{2}$ feet of the terminal ileum. In the others it was presumably due to the lack of nourishment during the first week following operation. On the other hand, of those who were given ether, numbering eight, there were two cases in which the immediate effect of the anaesthetic on the lungs was very obvious. In one there occurred quite a pulmonary oedema during the two days following the anaesthetic, which went on to the development of a serious pneumonic phthisis in the lower lobe of the patient's healthy lung, ending in death in the course of a few months. The advantage of gas-oxygen is obvious.

As regards wound healing, I find on the whole that it is very good, although not quite so good as in normal individuals. I have had in my own series of twenty-seven patients, only four disturbances of wound healing. One of these was aseptic, the wound breaking open for 3 inches on the eighth day under stress of coughing, and healing perfectly with secondary suture. One other resection case broke open for 3

inches to the peritoneum, partly from lack of healing, partly from slight infection. It healed also with no long delay with secondary suture. The other two cases were very slight infections in the skin, healing quickly. In the series of seventy odd tuberculous patients just mentioned, healing *per primam* has been remarkable by its regularity.

With regard to shock which, in beginning this series of serious major operations including so many resections and short-circuitings in patients who were often in poor general condition, I had expected to be severe, it has been remarkable by its absence. Only in two has there been the least cause for anxiety because of operative shock. These were both extensive resections in cases with advanced and slowly progressive pulmonary disease. In neither, however, was the shock sufficient to cause serious alarm, and was relieved by intravenous salt solution. I ascribe these good results to the excessive care which has been taken in keeping the patients warm; in allowing them nourishment up to shortly before the operation; in the skill with which the anaesthetic has been given, and the free use of local anaesthetic including in some cases infiltration of the root of the mesentery. These patients are undoubtedly fragile, and careless handling might break them. In addition I feel that part of the good results so far as the immediate effects of operation are concerned, have been due to the extremely favorable situation of our hospital which is high above the city on the slope of a hill and well provided with galleries on which the patients have been rolled out and have sometimes slept out.

As to the technique of operation, I have found it advisable in most cases to be prepared for exploration of the small bowel even where one can feel something in the caecum. Accordingly incision is usually made in the middle of the right rectus so that it can be enlarged up or down as necessity arises. The small bowel is always thoroughly explored beginning from the ileo caecal valve, peeling it out and returning it in order to avoid loss of heat, and counting the ulcers which are always marked on the peritoneal surface by a little patch of fibrinoplastic exudate, also measuring with sterilized tape approximately the length of bowel in which ulcers are found. In this way the whole of the ileum and jejunum can be perfectly well explored. It is important not to accept the apparent freedom of the lower foot or two of the ileum from disease as proof that there is no disease in the small bowel. It has happened on at least one occasion that a second operation showed ex-

tensive disease in the small bowel beginning nearly 2 feet above the valve and going upwards, when it was missed at the first operation.

As to the infection of the mesenteric glands it was remarkable how often they were only slightly enlarged, occasionally even being absent. It is rare to find them caseous at operation. Those which come conveniently within the area of resection are removed; but I have no hesitation in leaving some in the root of the mesentery when dissection would be difficult or occupy too much time. I have not found that they had any effect in later results. They are usually hyperplastic, and when the focus of disease is removed, may be expected to subside and become arrested.

The mortality of operation is surprisingly low. In the writer's series of thirty laparotomies in twenty-seven cases no operative death occurred. In the ten other cases collected from hospital records there was only one death from operation.

It might be urged *a priori* against operation in pulmonary cases that the post-operative distress would be apt to be excessive because of cough. In this I have been pleasurably disappointed. With the help of sedatives, it has been possible to get the patients through the first week with remarkably little pain; indeed with no more distress on the whole than is experienced by the ordinary abdominal case to whom ether is given.

In conclusion, while in the majority of cases of intestinal tuberculosis the lungs are already seriously diseased, and while in such cases our early hopes of bringing about arrest of the pulmonary condition by surgical removal of the diseased bowel have not on the whole been realized, it may still be emphasized that complete relief of bowel "misery" in these distressing cases may very often be accomplished by a well planned and carefully executed operation. The patients are keen for it, and are grateful after it. And then there remain the good cases, with but little pulmonary trouble and with eradicable bowel disease. These may frequently be cured by operation.

A summary of the results of operation is appended.

Results of operations for tuberculosis of the intestinal tract

EXTENT OF BOWEL LESION		PULMONARY LESION	OPERATION	RELIEF OF BOWEL DISTRESS		ULTIMATE FATE
				While in hospital	Later	
Appendix only	F.	None	Appendectomy	Complete	Complete	Normal health six years later
	G.	None	Appendectomy	Complete	Complete	Normal health two years later
	E.	Slight apical	Appendectomy	Complete	Complete	No abdominal trouble four years later. Chronic pulmonary tuberculosis
	B.	Slight apical	Appendectomy	Complete	Complete	Died two and a half years later from pulmonary tuberculosis
	R.	Chronic moderately advanced	Appendectomy	Complete	Complete	No abdominal trouble. Well six years later. Chronic pulmonary tuberculosis
	W.	Chronic moderately advanced	Appendectomy	Complete	Complete	Died four years later from pulmonary tuberculosis
			Palliative			
Appendix and caecum only or with ascending colon	W.	Bilateral, slight, active	Appendectomy only	Great	Partial	Alive, but with chronic pulmonary tuberculosis twenty months later
	S.	Bilateral, advanced, progressive	Appendectomy only	Great	Unknown	Died from pulmonary tuberculosis within one year
	A.	Bilateral, advanced, progressive	Appendectomy only	Great	Unknown	Died from pulmonary tuberculosis within one year
			Radical			
	S.	None	Resection: terminal ileum to transverse colon	Complete and striking	Complete	Well and working two years later
	E.	None	Resection: terminal ileum to transverse colon	Complete and striking	Complete till death	Died of acute obstruction seven months later
	M.	Advanced, chronic	Resection: terminal ileum to transverse colon	Complete and striking	Complete	Died over one year later from pulmonary tuberculosis: bowels uninvolved

Results of operations for tuberculosis of the intestinal tract—Continued

EXTENT OF BOWEL LESION		PULMONARY LESION	OPERATION	RELIEF OF BOWEL DISTRESS		ULTIMATE FATE
				While in hospital	Later	
Appendix and caecum only or with ascending colon	S.	Advanced, chronic	Resection: terminal to transverse colon	Complete and striking	Unknown	Died eight months later of brain lesion
	S.	Advanced, progressive	Resection: terminal to transverse colon	Complete and striking	Great, not perfect	Died sixteen months later of pulmonary tuberculosis
			Radical			
Caecum and rest of colon	T.	Bilateral, advanced, diffuse	Resection, with excision of sigmoid ulcer	Complete	Complete, striking till death	Died fifteen months later from repeated haemoptyses
	L.	Unilateral, advanced, progressive	Resection, ileo-transverse	Great	Unknown	Died about six months later from pulmonary condition aggravated by anaesthetic (ether)
			Palliative			
Both small and large bowel	H.	Moderately advanced, chronic	Appendectomy	Unknown	Unknown	Unknown
	M.	Moderately advanced, progressive	Appendectomy	Great for six months	Recurrence	Died a year and a half later from tuberculosis of lungs and bowels
	R.	Moderately advanced, chronic	Appendectomy	Great for one month	Unknown	Unknown
	C.	Bilateral, advanced, progressive	Appendectomy	Great till death	Great	Died three and a half months later, chiefly from pulmonary tuberculosis
	W.	Bilateral, advanced, progressive	Appendectomy	Partial	Unknown	Died in a few months of generalized tuberculosis
	M.	Bilateral, advanced, progressive	Appendectomy	Partial	Unknown	Died in a few months of generalized tuberculosis
	D.	Bilateral, advanced, progressive	Appendectomy	Great	Recurrence after four months	Died within a year of generalized tuberculosis

			Palliative			
	P.	Incipient, right apex	Ileo-sigmoidostomy	Partial	Complete	Alive and well two and a half years later: bowels and lungs in good condition
	S.	Bilateral, advanced, progressive	Ileo-sigmoidostomy	Striking for three months	Not so great	Died six months later, chiefly of pulmonary tuberculosis
	W.	Bilateral, advanced, progressive	Ileo-sigmoidostomy	Great for four months	Not so great	Died five months later of acute obstruction
	W.	Bilateral, advanced, progressive	Ileo-sigmoidostomy	Great (from pain) for two months	Not so great	Died three months later of generalized tuberculosis
			Radical			
Both small and large bowel	S.	None	Resection, ileo-caecal	Complete	Complete	Alive and working six years later
	G.	Bilateral, moderately advanced, chronic	Resection, ileo-transverse			Died three days after operation, probably from ileus
	C.	Bilateral, moderately advanced, chronic	Resection, three and a half feet, ileum to mid-transverse	Complete striking	Complete for eleven months	Died thirteen months after operation from ptomaine poisoning with aggravation of lung condition and dysentery
	J.	Bilateral, moderately advanced, arrested	Resection, five and a half feet, ileum to mid-transverse	Complete from pain, persistent diarrhoea	The same till death	Died in five months from chronic starvation and aggravation of lung disease, due to excessive exclusion
Colon and rectum	M.	None	Left artificial anus	Complete	Complete	Died one year later of shock following operation
	C.	None	Resection of caecum, right artificial anus	Complete	Complete	Alive and working three and a half years later: anus open

THE TREATMENT OF TUBERCULOUS HEMOPTYSIS WITH THROMBOPLASTIN AND EUGLOBULIN

GEORGE MANNHEIMER AND STANLEY L. WANG

From the Sea View Hospital

Hemoptysis occurs in 24 to 80 per cent of cases of pulmonary tuberculosis. So widely do statistics differ. In Sea View Hospital about 40 per cent of the patients have or have had hemoptysis. Probably less than one in a thousand consumptives die of hemorrhage. But the proportion of bleeding consumptives, who succumb to a hemorrhage, is naturally greater, about 2 per cent.

Hemoptysis is always an important event and renders the subsequent course of the disease rather doubtful. It always demands serious consideration. The fact that in most instances it stops spontaneously, makes the true valuation of remedial measures well-nigh impossible. This fact, however, does not justify therapeutic nihilism. On the contrary, it is our duty to try to limit the size and duration of a hemorrhage and thereby minimize its potential deleterious influence on the general and local condition. All reasonable measures must be employed. A certain routine prevails in all instances. In Sea View Hospital the bleeding person is put at perfect rest, receives the necessary nursing and help, ice to the heart or to the supposedly bleeding side of the chest, and ice by mouth. According to special indications, we use the following drugs: morphine hypodermically in selected cases, milder narcotics per os; magnesium sulphate and nitrites; sodium chloride by mouth or intravenously in hypertonic solution; and calcium salts. We have used horse serum hypodermically, and in a few instances whole blood from healthy human donors by intramuscular injection.

The remarkable hemostatic power of thromboplastin, when applied directly to bleeding surfaces, induced us to welcome the suggestions of Dr. A. F. Hess to use his preparation intravenously for tuberculous hemoptysis. We realized that the blood in the average tuberculous patient is not known to be deficient in substances necessary for coagulation. On the other hand we felt that we could do no harm by increasing those substances. Various measures which aim at contracting the

bleeding vessel either directly or reflexly or at lowering the blood pressure, act really by increasing the coagulability of the blood. Von den Velden of Duesseldorf has made elaborate studies on this subject and his various publications should be read by those interested.

The clotting of blood (that is the transformation of the fibrinogen of circulating blood into the insoluble fibrin of blood clot) has been shown to be due to the action of the fibrin ferment (thrombin) on the fibrinogen of the blood. The fibrin ferment or thrombin exists in the blood in the form of its forerunner (prothrombin) which is acted on by the calcium salts and converted into thrombin. Besides calcium salts, however, another factor is necessary. In spite of a sufficient supply of calcium salts, blood does not clot spontaneously within the vessels. This other factor may be furnished from the breaking down of blood cells or blood platelets or from the pieces of tissues that are cut or badly injured. It has been designated as "zymoplastic substance" by Schmidt, as "thrombokinase" by Morowitz, and as "thromboplastic substance" or "thromboplastin" by Howell. (*Amer. Jour. Physiol.*, 1912, xxxi, 1). Howell believes the reason that blood does not coagulate within the vessels is that the prothrombin exists there in combination with an antithrombin, which prevents it from acting. He believes that when blood is shed or flows over injured tissue the thromboplastin derived from blood cells, blood platelets or tissue cells combines with this antithrombin, liberating the prothrombin from combination with the latter. The prothrombin thus liberated now combines with the calcium salts, is converted into thrombin and converts the fibrinogen of the plasma into fibrin, causing coagulation to set in. Howell has shown that thromboplastin or "thromboplastic substances" from every source in which he has investigated it gives the reactions of the lipoid kephalin—the main lipoid of the brain. It is soluble in ether, but insoluble in water, alcohol and acetone, and kephalin prepared from the brain possesses the same action. (*Jour. A. M. A.*, August 5, 1916.)

Thromboplastin-Hess is prepared in the following manner: Ox brains are obtained fresh from the slaughter house, stripped of their membranes, washed in running water and weighed. They are then passed through a meat chopping machine three times, and an equal amount of normal salt solution is added. This suspension is allowed to remain in the refrigerator for forty-eight hours, and it is then twice pressed through cheese cloth. This extract, which contains a fine suspension of tissue in addition to tissue juice, is diluted with one-half its quantity of salt solution. Cresol is then added in proper proportion so that the finished preparation contains 0.3 per cent. It maintains its hemostatic potency

CASE NO.	SEX	AGE	STAGE	FEBRILE	DURATION	CONDITION	BED OR AMBULANT	PREVIOUS HEMOPTYSIS	PRESENT HEMOPTYSIS AND TREATMENT
1	F.	33	3	Hectic	7 years	Retrograding rapidly	Bed	Slight, 2 years ago	April, 1, 1916. Moderate, continued bleeding until April 10, 1916. 10 m. thromboplastin intravenously. No improvement. April 17, 1916, moderate continued. 10 m. thromboplastin intravenously. No improvement. June 5, 1916, Sputum heavily colored for past month, apparently continuous oozing. 20 m. thromboplastin intravenously at 1 p.m., 3 p.m., 5 p.m., and 7 p.m. Seemed improved on the following day, but on the next day the sputum became as heavily colored as before. June 10, 1916, sputum still heavily colored. 20 m. euglobulin subcutaneously. No effect
2	M.	55	3	Moderate	1 year	Retrograding slowly	Bed	Profuse April and May, 1915; Moderate, Aug., 1915; Slight, Dec., 1915; Profuse, June, 1916	August 2, 1916. Moderate; had occurred frequently for past two weeks, 10 m., euglobulin intravenously and 20 m., euglobulin subcutaneously at same time. No improvement. Rigor, malaise and nausea about 1 hour after injection, lasting about ¾ hour
3	F.	27	3	Slight	5 months	Retrograding slowly	Bed	Streaked, February, 1916	April 7, 1916. Moderate; then slight daily bleeding until April 10, 1916. 10 m., thromboplastin intravenously. No improvement. April 17, 1916, moderate. 10 m. thromboplastin intravenously. No improvement

4	M.	54	3	Slight	2 years 11 months	Retrograding slowly	Bed	Slight and Moderate, 6 times during past 2 years	April 6, 1916. Severe; then slight daily bleeding until April 10, 1916. 10 m. thromboplastin intravenously. No improvement. ✠ May 11
5	M.	37	2	Afebrile	1 year	Retrograding slowly	Ambulant	8 months ago, Streaked; 5 months ago, Moderate; and many Moderate since then at frequent intervals	April 4, 1916. Moderate; then slight daily bleeding until April 10, 1916. 10 m. thromboplastin intravenously. No improvement
6	F.	27	3	High, irregular	9 months	Retrograding rapidly	Bed	None	April 8, 1916. Moderate; repeated daily. Apparently moribund, April 10, 1916, 10 m. thromboplastin intravenously. No improvement. ✠ April 14
7	F.	17	3	Slight	1 year 2 months	Retrograding slowly	Bed	Severe, March 28, 1916, and April 15, 1916	April 26, 1916. Very profuse, nearly exsanguinated April 26, 1916. 20 m. thromboplastin intravenously. No improvement. ✠ May 5
8	F.	28	2	Afebrile	3 years 3 months	Retrograding slowly	Ambulant	Moderate and Severe frequently during past 3 years	April 23, 1916. Severe; then slight daily bleeding until April 24, 1916. 10 m. thromboplastin intravenously. No improvement. April 25, 1916, severe. 20 m. thromboplastin intravenously. No improvement

CASE NO.	SEX	AGE	STAGE	FEBRILE	DURATION	CONDITION	BED OR AMBULANT	PREVIOUS HEMOPTYSIS	PRESENT HEMOPTYSIS AND TREATMENT
9	M.	33	3	Moderate	8 months	Retrograding rapidly	Bed	Moderate, intermittent, for past 2 months	April 22, 1916. Moderate; then slight daily bleeding until April 25, 1916. 20 m. thromboplastin intravenously. Apparently improved. May 13, 1916, severe continued heavily streaked. May 17, 1916, 20 m. thromboplastin intravenously. No improvement. May 18, 1916, 20 m. thromboplastin intravenously. No improvement. ✠ July 16
10	M.	42	3	Moderate	5 years	Retrograding rapidly	Bed	Profuse August, 1915	April 24, 1916. Profuse; then moderate until April 26, 1916. 20 m. thromboplastin intravenously. No improvement. Continued moderate daily. 20 m. thromboplastin intravenously daily in a.m., May 5, 6, 7, 8 and 9, 1916. No improvement. May 19, 1916, continued moderate daily. 20 m. euglobulin intravenously. No improvement. Slight chill a short time after injection. ✠ August 26
11	M.	55	3	Afebrile	1 year	Retrograding slowly	Ambulant, until May 9, 1916	Profuse, April 27, 1915, and May 27, 1915; Moderate, Aug. 11, 1915; Slight Dec. 9, 1915	May 9, 1916. Profuse; then heavily streaked daily; May 13, severe; then slight until May 17, 1916. 20 m. thromboplastin intravenously. No improvement. May 18, 1916, 20 m. thromboplastin intravenously. No improvement. Many hemorrhages until death September 28.

12	M.	25	3	Slight	1 year	Retrograding slowly	Bed	Streaked, May 22, 1915; Profuse, June 22, 1915. Past 5½ months moderate, about every six weeks	May 17, 1916. Moderate; then heavily streaked daily. May 22, 1916, 20 m. euglobulin intravenously. No improvement. Macular eruption, apparently serum rash appeared a few hours after injection. ✠ July 6
13	M.	27	3	Moderate	6 months	Retrograding rapidly	Bed	Streaked, December 20, 1915; Slight, March 27, 1916; Moderate, April 3, 1916	May 22, 1916. Sputum heavily streaked daily following artificial pneumothorax. 20 m. euglobulin intravenously. No improvement. Slight chill few hours after injection
14	M.	34	3	Moderate	5 months	Retrograding slowly	Bed	Slight, March 28, 1916	May 23, 24, 1916. Profuse. May 24, 1916, 20 m. euglobulin intravenously. No improvement. Chill about 1 hour after injection, increased temperature and felt very uncomfortable. Hemoptysis seemed increased
15	F.	29	3	Moderate	3 months	Retrograding rapidly	Bed	Slight, March 15, 1916	June 6, 1916. Profuse at 10.30 a.m. 20 m. thromboplastin intravenously at 3 p.m., 5 p.m. and 7 p.m. No improvement. June 7, 1916, 20 m. euglobulin subcutaneously. No improvement. Temperature increased temporarily. ✠ July 6

CASE NO.	SEX	AGE	STAGE	FEBRILE	DURATION	CONDITION	BED OR AMBULANT	PREVIOUS HEMOPTYSIS	PRESENT HEMOPTYSIS AND TREATMENT
16	M.	32	3	Slight	1 year 3 months	Retrograding slowly	Ambulant	Slight, May 31, 1915; Profuse, Nov. 14, 1915; Moderate, March 13, 1916	June 13, 14, 1916. Profuse. 20 m. Thromboplastin intravenously at 11 a.m., 1 p.m. and 3 p.m. No improvement. June 15, 1916, 20 m. euglobulin, intravenously. No improvement
17	F.	50	2	Afebrile	1 year 2 months.	Retrograding slowly	Ambulant	Streaked four times during past year	June 25, 26, 1916. Profuse. 20 m. thromboplastin intravenously at 1 p.m., 3 p.m., 5 p.m., 7 p.m. and 9 p.m. No improvement. June 27, 1916, 20 m. euglobulin subcutaneously. No improvement. Slight chill 4 hours after injection
18	F.	27	3	High	3 months	Retrograding rapidly	Bed	None	June 20, 21, 22, 1916. Profuse. 20 m. thromboplastin intravenously at 11 a.m., 1 p.m., 3 p.m., 5 p.m. and 7 p.m. No improvement. June 23, 1916, 20 m. euglobulin subcutaneously. No improvement. ✠ July 12
19	F.	19	3	Moderate	4 months	Retorgrading rapidly	Bed	None	June 16, 1916. Profuse; then daily moderate bleeding until June 21, 1916. 20 m. thromboplastin intravenously at 3 p.m., 5 p.m. and 7 p.m. No improvement. June 22, 1916, 20 m. euglobulin subcutaneously. No improvement

20	F.	23	3	Moderate	4 months	Retrograding slowly	Bed	None	August 1, 1916. Moderate. 10 m. euglobulin intravenously and 20 m. euglobulin subcutaneously at same time. No improvement
21	F.	26	2	Slight	11 months	Retrograding slowly	Ambulant	Streaked September, 1915; Profuse December, 1915	July 17, 1916, heavily streaked continued daily. July 22, 1916, 10 m. euglobulin intravenously and July 23, 1916, 20 m. euglobulin subcutaneously. No improvement
22	M.	31	3	Moderate	2 years 8 months	Retrograding slowly	Bed	Profuse January, 1914; Streaked September, 1915	September 3, 1916. Profuse; then slight daily with occasional profuse exacerbation. September 21, 1916, 30 m. euglobulin subcutaneously. No improvement. Slight malaise for about 24 hours after injection
23	F.	27	2	Slight	2 months	Retrograding slowly	Ambulant	Moderate, June, 1916	August 3, 1916. Profuse. 10 m. euglobulin intravenously and 20 m. euglobulin subcutaneously at the same time. No improvement. Chill and general malaise for 3 or 4 hours after injections

for some time (several weeks). For internal use a 1 per cent solution is employed.

Euglobulin is a normal protein of the blood serum. It represents the styptic element and has much less anaphylactic action than the serum albumin.

Both preparations were kindly supplied to us from the research laboratory of our health department by Dr. A. F. Hess, to whom we feel greatly indebted.

Each dose of thromboplastin was boiled before usage. In all but one case it was injected intravenously. The euglobulin was administered both subcutaneously and intravenously.

We selected cases with moderate or copius hemorrhages which had a tendency to continue. The terms are those of Lawrason Brown as given in Osler and McCrae's *System of Medicine,* 1913 edition, namely: Streaky; slight (less than 30 m.); moderate (30 to 100 m.); severe or copious (100 to 250 m.); profuse (over 250 m.). These amounts refer to the quantity of blood lost in twenty-four hours.

Twenty-three cases were treated. All had definite pulmonary tuberculosis with positive sputum. Other medicinal treatment was omitted for the time being.

In the beginning small doses were used, as nothing was known about the effects. They were subsequently increased and also given more frequently. When a hemoptysis recurred in the same individual at a later period different dosages were administered. This refers only to thromboplastin, which was used in 23 instances as follows:

In nine cases 10 mils were given; in four, 20 mils; in two, 2 doses of 20 mils each on consecutive days; in one, 5 doses of 20 mils each on consecutive days; in three, 20 mils every two hours for 3 consecutive doses; in one, 20 mils every two hours for four consecutive doses. No bad effects or reaction of any kind resulted.

In cases 1 and 9 a hemorrhage ceased after the injection of thromboplastin, but recurred subsequently and was not influenced by a repetition or increase of the dose. A causal connection can therefore not be assumed. In all the other cases there was even no apparent benefit.

Euglobulin was administered 15 times in doses varying from 10 to 30 mils subcutaneously and intravenously. The idea was to supply hemostatic material for immediate absorption and to create a depot from which it could gradually be absorbed to make the effect more lasting. There was no improvement in any case. A few patients showed slight re-

actions as evidenced by a chill, elevation of temperature and general malaise. One developed a typical serum rash. These reactions were not severe and were of short duration.

The series is small and allows of no definite conclusions. Modifications might be tried or combinations with other measures, such as administration of salt or tying the limbs.

One of us (Mannheimer) has used the latter measure successfully in private practice and intends to use it systematically in the hospital. Larger doses might be necessary. Dr. Hess tells us that he has introduced 180 mils of thromboplastin into the veins of an eight-year-old hemophilic girl. Stronger dilutions might be tried, acting on the findings of Hess, that the injection of 2 mils of 10 per cent thromboplastin into the veins of a rabbit shortened the coagulation time one-half. But, of course, there is a limit to the concentration of the solution. One of us (Wang) at the suggestion of Dr. E. S. McSweeny, injected 5 m. of undiluted thromboplastin, such as is used for local application, into the ear vein of a rabbit. The rabbit died almost immediately and a large firm blood clot was found in the right auricle, which extended downward into the right ventricle and upward into the veins of the neck.

On the whole it seems more reasonable to use larger quantities of a weak solution than small doses of a strong one.

Altogether our results with thromboplastin and euglobulin in the treatment of tuberculous hemoptysis have been negative.

TUBERCULOSIS AND PREGNANCY[1]

A STUDY OF THREE HUNDRED CASES WITH A REVIEW OF THE LITERATURE

S. A. DOUGLASS AND J. E. J. HARRIS

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In the analysis of the histories of a series of tuberculous women admitted to the Ohio State Sanatorium from December, 1909, to April, 1917, inclusive, it is apparent that in a large number of cases we are dealing with an exacerbation of a latent or quiescent tuberculosis, rather than with an initial lesion, and that in married women pregnancy is one of the leading factors in lighting up this inactive process.

Under the term "pregnancy" we have included the period from conception to the end of the puerperium. To determine its importance as an etiological factor, 392 married tuberculous women were considered, of whom 92 gave no history of pregnancy, leaving 300 cases in the series on which to base our observations. In 88 of these cases, or 29 per cent, pregnancy was the leading factor to which the onset of tuberculosis was attributed. In 24 additional cases, or 8 per cent, pregnancy was a presumptive factor, making a total of 37 per cent.

Turban, from Schauta's clinic, reports 29 per cent of tuberculous women giving a positive history of the disease originating or becoming definitely recognizable during pregnancy or the puerperium. The combined statistics of Fishberg, Funk, Jacob and Pannwitz, and a number of other observers, comprising in all a series of more than 1100 cases show that in 39 per cent of these cases the active symptoms originated during pregnancy or the puerperium. Scarborough reports 94 of 200 married women admitted to the Iowa State Sanatorium, or 47 per cent, in whom symptoms of activity appeared during or after childbirth, while Trembley of Saranac Lake states that of 200 married tuberculous women 63 per cent gave a similar history.

From these statistics we can safely say that pregnancy is a predominant factor in the production of an active tuberculosis in approximately

[1] Read before the clinical section of the thirteenth annual meeting of the National Association for the Study and Prevention of Tuberculosis, Cincinnati, Ohio, May 10, 1917.

40 to 45 per cent of married tuberculous women. Other observers believe that it plays a more important rôle: thus, exacerbation of tuberculosis in pregnancy, birth and child bed was noted by Felner in 68.3 per cent of his cases; by Kaminer in 80 per cent; by Pradella in 90 per cent; by Pankow and Kupferle in 94.5 per cent and by Rosthorn in 100 per cent.

The different percentages may be explained by the stage of the disease present or by its activity. Norris reports 68 cases, in whom active symptoms were observed in 20 per cent of the mild, quiescent cases, and in 70 per cent of the more advanced cases.

In our series the disease proved fatal in 30 per cent of the cases within eighteen months. Bacon states that 33 per cent of pregnant tuberculous women die within a year of their labor. Lobenstine reports a fatal termination in 40 per cent of von Bardeleben's cases, and a fatal termination within two months in 50 per cent of the severe cases in his series. Fifty-three per cent of the cases reported by Trembley are dead, while Schottelius and Schauta's statistics show a mortality of 75 per cent.

There are numerous factors responsible for the aggravation of tuberculosis during pregnancy and the puerperium. Pregnancy makes increased demands on metabolism, oxygenation and innervation, on circulation and elimination. The point of least resistance, in these cases a quiescent pulmonary lesion, is put to the test and the latent tuberculous focus breaks down. The presence of the gravid uterus interferes with respiration and the aeration of the blood; the nausea and vomiting of pregnancy tend to interfere with assimilation; the prolonged muscular exertion of labor, the loss of blood and its attending exhaustion, the use of the anaesthetic, auto-infection by the aspiration of infected material from an old focus in the lung into healthy portions, and the possibility of toxins being thrown into the system from the placental site are all serious factors. In addition to these factors we have in most cases the strain of subsequent lactation, and the responsibility incident to the care of the child.

Occasionally we see that during the pregnant state the tuberculous process apparently improves and the general condition of the patient is strikingly ameliorated. This is true, however, only of the fibroid type, which, it has been observed, is hardly modified at all either during gestation or after delivery. This is responsible for the traditional view held until recent times that pregnancy may sometimes arrest tuberculosis, and because of this belief writers of former generations, like Cullen,

recommended marriage to tuberculous women. Grissole, however, in 1849 published an article of such force that it shook the dogma which until then had remained firm. Townsend, in 1897, speaking of cases observed at the Boston Lying-In-Hospital, says that, "During pregnancy the patient often seems better, and the disease appears in abeyance, and nature seems to put forth a supreme effort to suppress the disease during pregnancy and to make the labor easy and short, but after the child is born the disease advances at a rapid rate."

Experience shows that in 40 to 45 per cent of married tuberculous women we can trace the onset of active symptoms to pregnancy. This brings up the important question of prophylaxis. Many approve the dictum of Auvard, as applied to the tuberculous, when he states, "The young girl should not marry—the married woman should not conceive—the mother should not nurse her child." We believe, however, that it is impractical to formulate so general a ruling because in a considerable percentage of cases pregnancy has little or no influence on the tuberculous process. Many of our discharged patients, who have had no clinical evidence of activity for a period of one to five years, have gone through childbirth with no renewal of their disease. Just what influence sanatorium education and training may have had in forefending these arrested cases against relapse during their pregnancies is hard to estimate. It is to be expected however that they have been under rather good conditions, many of them under ideal conditions, and that they have applied their knowledge of a hygienic-dietetic regimen throughout this period.

As the tuberculous mother runs a very great risk of having her old tuberculous lesion reactivated, and as she brings into the world an individual who is almost destined to become tuberculous, it readily becomes apparent that the question of therapeutic abortion is a constant issue in the practice of tuberculosis and obstetrics. There is, however, no general agreement as to the policy which should be pursued in dealing with this problem. The moral aspect of interfering with pregnancy is a grave subject. It cannot be denied that such a custom might readily become a source of abuse by furnishing a pretext for malpractice. Each case must be considered upon the merits of its several features. With manifest active tuberculosis, pregnancy should be avoided. If conception has taken place, therapeutic abortion is indicated when the lesion is early or moderately advanced.

Three schools or groups as described by Schauta have come into

existence in so far as abortion in tuberculosis is concerned. The first school says, "Abort every women that has tuberculosis." The second school says, "Abort no woman that has tuberculosis, give her the best of treatment and let her go through her pregnancy." The third school holds that, "every case must be treated individually." It seems to us that the latter is the only rational view and the only course to pursue.

The two important indications for terminating pregnancy are vital and prophylactic. Inducing abortion in an active advanced case to save life is of little value, as most authorities agree that advanced cases decline as rapidly after the operation as without it, and if a woman is in such condition that she is pretty sure to die if she goes on with her pregnancy, she will be almost certain to die after abortion is induced. Where a case is seen after the fifth month of gestation interference is not deemed advisable and expectant treatment should be the rule as practically all agree that interference at this stage is of little value.

Knopf, in discussing Lobenstine's paper read before the New York Academy of Medicine, in November, 1912, said that heretofore he had assumed the attitude of non-interference in early and moderately advanced tuberculosis cases of pregnancy, but he was willing to confess that he was more and more inclined to the emptying of the uterus in all cases where a positive healed tuberculous condition existed. It appears to us that this view is perhaps too radical although Knopf's opinion is also held by Fishberg.

Where the lesion is an active one, immediate aborn stosi indicated in the early or moderately advanced types. In the preience of an extensive lesion, in the quiescent state, the pregnancy should be interrupted if the patient is seen before the fifth month. Another indication for the termination of pregnancy is the appearance of a tuberculous lesion in some other part of the body, especially laryngeal tuberculosis. Of 97 cases reported in the literature where laryngeal tuberculosis complicated pregnancy, 90 have died.

The prophylactic indication lies in the prevention of childbirth in a woman who has a good prospect of success in her fight against tuberculolosis. Interruption of pregnancy before the fifth month in early cases leads to definite improvement in 65 to 70 per cent, provided suitable treatment is applied (Norris). Before deciding on this indication it is necessary to consider the duration of the pregnancy, the character of the pulmonary lesion, and the social status of the patient. Where the pulmonary lesion is early or moderately advanced and the process

remains quiescent and the symptoms of pregnancy are not severe, indications for interference or non-interference depend on modifying circumstances. If the patient can be properly treated at home or in a hospital, the labor properly conducted, and the child taken from the mother immediately and properly cared for, our duty is to avoid interference. The more prominent factors which should influence our decision in these cases are the patient's ability and willingness to undergo proper hygienic-dietetic treatment, and the question of whether she already has one or more children. Bacon states that the indications for the induction of abortion grow less and less as we improve our management.

Nursing of an infant by a tuberculous woman has a double menace. Even though the mother seems to be in fairly satisfactory condition, her ultimate recovery is more likely to be retarded if nursing is indulged in and the child is almost sure to get a massive infection. In all active cases the only rational measure is prompt isolation of the child. The danger of the close association is self evident. The possibility of direct infection through the milk must be taken into account. It has been shown that in exceptional cases human breast milk contains tubercle bacilli. Deutsch believes that the milk of tuberculous mothers carries toxic products which reduce the resistance of the infant. Clinical experience shows that a large percentage of infected children are those who had been nursed by their mothers. When we add to these cases the vast army of children who later develop the disease from exposure and contact, the enormous toll on health and life due to faulty inheritance and faulty surroundings can be appreciated. The interests of both mother and child are best served by beginning with a wet nurse or artificial feeding immediately following birth.

In conclusion: the tuberculous married woman should be instructed concerning the danger of pregnancy as it is an important factor in lighting up a quiescent tuberculosis. Pregnancy should be avoided until the pulmonary lesion has been arrested for several years. When conception has taken place, pregnancy should be terminated before the fifth month in all active cases of early and moderately advanced pulmonary tuberculosis, and in all cases of advanced tuberculosis when the process is quiescent.

It is for the best interest of both mother and child to prohibit the tuberculous mother from nursing her child.

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SOME EXPERIMENTS WITH TUBERCULOUS SPUTUM TWENTY-TWO YEARS OLD

NEWELL BLY BURNS

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The experiments were performed with material said to have been sputum obtained from a tuberculous patient in the year 1895.

Through the kind thoughtfulness of Mr. Seymour Stone, Secretary of the Boston Anti-Tuberculosis Society, and Dr. A. K. Stone, Chairman of the Massachusetts State Commission on Tuberculosis, the material came into my hands. The opportunity thus provided for carrying out this unique investigation I thoroughly appreciate.

In November, 1916, we received at the laboratory of the North Reading State Sanatorium a small box in which were snugly packed three 4-ounce wide-mouth bottles. The bottles were tightly corked.

The wooden box was evidently the original receptacle in which the bottles were packed, for the packing material consisted of newspaper, copies of the New York *Herald* for Thursday, May 16, 1895. Two such headings were found on the newspaper wrappings about the bottles. It was quite evident that the bottles had lain undisturbed in this box for a long period.

Each bottle was labelled with the same patient's name. I numbered the bottles; and the contents, according to number, were:

1. A dark brown perfectly dry crusty substance covering evenly and firmly adherent to the bottom to the depth of one-half inch.

2. Perhaps a half-ounce of thick liquid, dark brown, and of an oily appearance.

3. About an ounce of thick opaque fluid, resembling somewhat coffee grounds, and dark brown in color. Some sediment.

I have sought to demonstrate:

1. The presence in this material of organisms resembling tubercle bacilli.

2. The viability of such tubercle bacilli as might still exist.

EXPERIMENTS WITH MATERIAL IN BOTTLE 1

With due precautions against contaminating the contents the cork stopper was withdrawn. It crumbled considerably in this proceeding owing to its dry condition. However, it had effectively sealed the bottle.

With sterile forceps I attempted to detach a fragment of the dry crust from the bottom of the bottle but found the substance too hard.

I then added 40 cc. sterile salt solution, 0.8 per cent, to the bottle and ran it in the shaking machine for fifteen minutes. The solid material was dissolved to a certain extent although there was considerable sediment after the bottle had stood for a few minutes.

Smears were prepared on glass slides from: (1) the foamy top layer of the liquid; (2) the middle of the liquid (by pipette); (3) the sediment.

The smears were stained by the carbol fuchsin—Gabbett's methylene blue method.

All smears showed the presence of acid fast bacilli in a mass of detritus. The detritus was of a non-recognizable character. In the smear from the sediment the organisms were especially numerous.

The red staining bacilli were beaded, curved, mostly short and some apparently fragmented. The outlines were roughened but the beaded refractile character was as characteristic as though seen in a smear from a fresh culture.

Culture media were inoculated with several loopfuls of the solution as follows: (1) Blood serum slant; (2) glycerin bouillon; (3) glycerin potato extract slant; (4) plain bouillon.

No growths resulted.

Animal inoculation. March 1, 1917. Guinea-pig 72, about 450 grams in weight, was inoculated intraperitoneally with 1 cc. solution prepared as above described, so that four parts represented one of solid substance; and 0.5 cc. of the same solution was inoculated intramuscularly into the right leg.

March 2, 1917. Guinea-pig 73, about 500 grams, inoculated with sediment of bottle No. 1 reshaken with sterile salt solution, 2 cc. subcutaneously in groin and 0.7 cc. in left axilla.

March 8, 1917. Guinea-pig 74, about 500 grams, was inoculated with similar material as used in pig 73 except that the material was incubated six days. Intraperitoneally 2 cc., and subcutaneously, left axilla, 1 cc. were inoculated.

April 17, 1917. Guinea-pig 72 found dead. Necropsy showed animal poorly nourished with no palpable glands and no signs of abscess in the right leg or in abdomen. Abdominal wall was thin and showed very little fat. No infiltrated or caseous glands noted. Spleen small, liver and lungs apparently normal. All smears negative for acid fast bacilli.

March 8, 1917. Guinea-pig 75 inoculated as a control with sterile salt solution such as was used in making dilutions in bottle 1.

June 22, 1917. Guinea-pig 73 living and apparently well.

June 22, 1917. Guinea-pig 74 living and apparently well.

June 25, 1917. Guinea-pig 75 living and apparently well. No signs of tuberculosis.

Necropsy. June 25, 1917. Guinea-pig 73. Animal had gained in weight and was well nourished. Nothing at points of inoculation. Peritoneum, liver and spleen normal. No enlarged, infiltrated or caseous glands found. Lungs show congestion, especially the right and are bloody on section, but do not sink in water.

Necropsy. June 25, 1917. Guinea-pig 74. Animal well nourished. Peritoneum, liver, spleen and lungs normal. No glands. No excess of fluid in abdomen. Nothing noted at points of inoculation.

EXPERIMENTS WITH MATERIAL IN BOTTLE 2

The stopper on withdrawal left fragments attached to the mouth of the bottle and crumbled somewhat, some of the fragments falling into the material.

Upon dipping the platinum loop in the material small stringy semi-mucoid masses were engaged, although the bulk of the material was perfectly liquid.

It was, however, difficult to make a smear stick to the glass slide during the staining process.

By the use of antiformin a good smear stained with carbol fuchsin and Gabbett's methylene blue solution was obtained. The antiformin sediment had been washed in distilled water and precautions were taken to prevent the accidental introduction of acid-fast bacilli.

Microscopical examination of this smear revealed scattered acid-fast bacilli, beaded, some curved, some apparently fragmented, and refractile. The organisms looked frazzled with their roughened outlines and in a state of desiccation.

On culture media, agar slant, a faint watery gelatinous streak appeared on the fourth day. This growth was in pure culture and when

stained with methylene blue revealed a very large, long and broad bacillus. It was non-acid-fast.

An agar stab developed in twenty-four hours a vacuole in the media suggesting that the organism was of the gas forming variety. No further attempt was made to identify this germ.

Animal inoculation. December 13, 1916. Guinea-pig 69 was inoculated with 1 cc. solution made up of 0.5 cc. material and 0.5 cc. sterile salt solution.

This pig continued to be strong and healthy and showed no signs of disease.

Necropsy. June 25, 1917. Guinea-pig 69. Animal well nourished. Nothing at points of inoculation. Peritoneum, spleen, liver and lungs were normal.

EXPERIMENTS WITH MATERIAL IN BOTTLE 3

The contents, upon agitation with the platinum loop seemed to be of a stringy semi-mucoid character with some brownish dirty yellow sediment. The reaction was neutral. There was a disagreeable, foul odor.

This specimen was treated in the shaking machine in its original container and a homogeneous, yellowish brown frothy liquid resulted. Some sedimentation gradually occurred.

Several smears, taken directly from this material, showed acid-fast beaded organisms of the same appearance and frazzled condition as those noted in the foregoing material. One imagined that these organisms were undergoing some slow process of disintegration.

The background of the smears was a mass of detritus containing occasional non-acid-fast bacillus-like bacteria which took the blue counter-stain.

All culture media inoculated were soon overgrown with thick, almost colorless growths.

Microscopically this early growth was seen in pure culture to consist of thick short bacilli, not acid-fast, some with vacuolated ends and many tending to form chains.

Older growths, stained with methylene blue, showed masses of blue staining branching threads, crisscrossed with shorter and broken forms dotted with more deeply staining granules.

Animal inoculation. February 23, 1917. Guinea-pig 70, of about 500 grams in weight, was inoculated with the undiluted specimen freshly shaken. Intramuscularly 0.5 cc. was injected in the right thigh and subcutaneously 0.5 cc. in the right groin.

February 23, 1917. Guinea-pig 71, of about 500 grams in weight, was inoculated with the undiluted freshly shaken specimen, 1 cc. intraperitoneally and 1 cc. intramuscularly in the left shoulder.

Both pigs continued to gain weight and remained in healthy and lively condition.

Necropsy, June 25, 1917. Guinea-pig 70. Animal well nourished and fatter than at time of inoculation. No glands, no fluid in abdomen. Peritoneum, liver, spleen, lungs were normal. Evidently a healthy animal. Nothing at points of inoculation.

June 25, 1917. Guinea-pig 71. Animal apparently normal. Weight increased. Abdomen normal. Peritoneum, spleen, liver and lungs negative. Nothing at points of inoculation.

RESULTS DEMONSTRATED

1. In all the material acid-fast organisms resembling tubercle bacilli structurally and in staining qualities were seen to be present. They were more numerous in the dried material in which no other organism could be found.

2. No growth was obtained such as would prove, or even suggest, that these organisms still retained their viability. Inoculation of both culture media and guinea-pigs produced only negative results in the attempts to recover living tubercle bacilli from the material examined.

Some work of this sort was carried out by Dr. A. K. Stone (1) of Boston. Dr. Stone examined some tuberculous sputum which was set aside three years before. "The bacilli took the stain beautifully and appeared as bright red as though they had been stained from a fresh specimen." Having shown the presence of bacilli in sputum kept for three years he proceeded to prove their virulence and was successful. Dr. Stone used rabbits for inoculation. He concluded that "the bacillus of tuberculosis when three years old was not only capable of receiving staining material, even though the medium in which they were suspended had been reduced for a period of more than two years to absolute dryness, but that it was also surely alive and virulent."

I have found no other reference in the literature relative to the preservation of tubercle bacilli in sputum three or more years old.

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(1) STONE, A. K.: Why the sputa of tuberculous patients should be destroyed. Am. Jour. Med. Sc., March, 1891.

CLINICAL OBSERVATIONS DERIVED FROM THE EXAMINATION OF OVER THREE THOUSAND CHESTS CHECKED BY STEREOROENTGENOGRAMS[1]

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I have previously proved that a definite X-ray marking characteristic of tuberculosis exists upon the stereoroentgenogram in cases over twelve years of age, suffering from pulmonary tuberculosis, and that this marking definitely differentiates the lesion of tuberculosis from the normal.

What I wish to present to you now are some clinical observations derived from the study of more than three thousand cases, examined both clinically and by the stereoroentgenogram. There is no originality in these observations, but there is much emphasis, and I hope some value in their selection and repetition.

This coördinated examination gives us our best chance of obtaining an understanding of the pathology of pulmonary tuberculosis in the living, and greatly facilitates the study of the pathology of the lung when removed from the thorax. This knowledge of the pathology of pulmonary tuberculosis enables us to interpret physical findings and it is as though we were making our physical examinations always under a great master. Thus, we are able to direct more definitely the researches of the clinical laboratories and our physical examinations than would be otherwise possible. Also the prognosis can be made with a degree of certainty that affords the greatest satisfaction and protection to the clinician, the patient, and his family.

Having these pathological conditions clearly before us, the rationale of treatment and the fallacy of specifics are so emphasized as to preclude false hopes in the Friedmann fraud and similar fallacies, and to necessitate the discipline and self control that form the only "sure cure." For we are enabled to determine exactly to what stage the disease in

[1] Read before the clinical section of the thirteenth annual meeting of the National Association for the Study and Prevention of Tuberculosis, Cincinnati, Ohio, May 10, 1917.

each patient has progressed, and are not forced to wait for the ravaging fevers or lung destruction as evidenced by elastic tissue and tubercle bacilli, before putting the patients upon an adequate rest schedule. The certainty with which deductions can be drawn from the coördinated physical and clinical findings approximates the work of the average doctor to that of the great clinician.

Then too, the study of these X-ray plates makes possible and imperative surgical intervention when needed in diseases of the thorax, to an extent never previously encompassed except by the few master minds and hands.

Finally this scientific study of absolute densities as applied to diseases of the thorax tears the last veil from before that accurate mental picture, that it is so necessary to see clearly before daring to apply the art of medicine in this field.

It is interesting to note how closely the findings from a study of roentgen plates conform to the pathological findings as seen at the autopsy. The belief is common that the X-ray plate is a study of the progress of the disease, but that the post mortem findings are simply end results. To illustrate that the two studies really give us similar results consider the constant witness of both to the facts of the greater involvement of the apices in the adult and that the lung margins at the base are the last to be involved, whereas in children early basal involvement is much more common. Again they both show that the disease progresses from above downward, so that the apices of the lower lobes are frequently involved before the lower part of the upper lobes. However, the X-ray accentuates the fact, often not accentuated by the pathologist, that this progress early in the disease generally lacks continuity, healthy areas being found between tuberculous lesions. Pathologists have generally conceded that the apex of the right upper lobe is much more frequently involved than that of the left, clinicians often claiming a 75 to 25 percentage. Our X-ray studies would tend to greatly modify this, and show that the percentage is not more than 55 to 45. The value of rectifying this error becomes apparent in our physical examinations, when we remember that it is more difficult to detect slight pathological changes in the upper left than in the upper right lobe.

The extent to which the thoracic viscera may be dislocated by the scar tissue resulting from tuberculosis is almost beyond belief, unless the subject has been given especial consideration. The upper lobe is

often foreshortened and the middle or lower lobes expanded by emphysema to fill the thoracic cavity. This might easily lead to the clinical mistake of thinking that you have the involvement of only half of an upper lobe, when in reality the entire lobe is destroyed. Such mistakes can be avoided by the X-ray examination. The heart and vessels are frequently dislocated upward and it is well to remember that this may be the cause of accentuated aortic and pulmonic sounds. A lateral dislocation of the heart frequently accounts for a systolic murmur. Often the heart is drawn entirely into the right or left thorax. At such times the ribs on that side are contracted. When this occurs to such an extent that this side is immobile while the other side expands freely, it is easy to predict the position of the heart from inspecting the back. The trachea may be drawn so far to either side that a large cavity is suspected at one or the other apex, where none exists. It is often impossible to correctly outline the heart by percussion in a case of fibroid tuberculosis. I do not mean that this can never be done, but that one is so often mistaken that physical findings are always in doubt.

The stereoroentgenogram is of great value in diagnosing hemorrhagic infarct. Six of these cases have been studied. All of them were referred for diagnosis of tuberculosis because of hemorrhage. The lesion in each case was within the upper half of the right or left lower lobe. None of them came to autopsy and thus the diagnosis may be questioned. The diagnosis however was based upon hemorrhage with complete recovery, no tubercle bacilli found in the sputum, no evidence of tuberculosis upon the X-ray plate, but a homogeneous cone-like density over the area, which showed loss of resonance, distant breathing and adventitious sounds. The location of these infarcts near the apex of the lower lobes, does not agree with the pathological reports of Professor Woolley of the Cincinnati General Hospital, kindly placed at my disposal by Professor Morris. Twenty-five infarcts were definitely located, two in the upper right, two in the upper left, five in the middle lobe, four in the upper part of the lower right lobe and four in the lower parts of lower right lobe, two in the upper part and six in other parts of lower left lobe. My X-ray studies would indicate that hemorrhage can be due to infarct of the lung without tuberculosis and that the apices of the lower lobe should receive most careful examination. While Professor Woolley's cases show only six out of twenty-five infarcts to be in the apices of the lower lobes, it must be noted that their clinical condition was entirely different from those of my series. My

cases were apparently in good condition, as though suffering from incipient tuberculosis, while his were at the end of life, suffering from syphilis, septicaemia, nephritis, etc.

There is no more interesting or instructive X-ray study than that of pleural effusion. As a general proposition it can be stated that pleural effusion does not move and that movable dulness over the thorax means hydro-pneumothorax. Let us examine the exceptions to this. Pleural effusion will move after it has been tapped. Professor William S. Thayer has told me that he has known pleural effusion to move when there was an old emphysema in the lung above it. Professor Roger Morris teaches that transudates such as are due to heart disease will move. I cannot corroborate this though I have no reason to doubt it. I have seen one case in a child with cavities in the lung apparently not due to tuberculosis in which the pleural effusion moved. I have examined thirteen cases of lung tumor with effusion which came to autopsy. Eleven were carcinoma and two were sarcoma. In each of these cases the pleural effusion moved slowly in marked contrast to the quick changes of dulness in hydro-pneumothorax. In more than one hundred cases the pleural effusion was associated with tuberculosis of the lungs and it could be demonstrated by the use of the roentgen rays not to move as much as a half inch.

If we study these facts we have pleural effusion with stable dulness, lung tumors with slowly movable dulness and hydro-pneumothorax with freely movable dulness. From these we must differentiate emphysema with effusion, transudate, and a few rare conditions such as the child with multiple non-tuberculous cavities. If we can say that an effusion which causes a slowly movable dulness is probably due to lung tumor we have a very valuable clinical sign. To do so all that is necessary is to examine for emphysema, and to ascertain if the heart or kidneys could account for the effusion.

I believe that it is a fact that a slowly movable dulness which cannot otherwise be accounted for should always cause the clinician to suspect lung tumor which is probably malignant. The X-ray again points out what the clinician should have learned from the pathologist, that lung tumors are quite common. Pleural effusion in bed patients, accumulated during a recumbent position, rises much higher in the back than that in ambulatory patients.

Pleural effusion is best diagnosed by the X-ray. Its extent, its surface, whether or not encapsulated. the amount of lung compressed,

the displacement of the heart, are told with accuracy. Nine times out of ten it is easy to diagnose clinically but the tenth time the physical signs will foil the élite. And I want to say here that the careless and bold use of the aspirating needle should be considered a crime. I have known of many cases where abscess, gangrene and septicaemia have followed aspiration and I am sure that many other fatal results have been avoided by the resistance of the patients' systems in spite of the mistakes of the operator.

Pleural effusion if not distressing should be let alone, because more damage than good may result from interference. If distressing, it should be tapped, and as much fluid removed as can be taken without difficulty. The lung should then be collapsed by the injection of gas. This lightens the weight in the thorax, reduces the amount of fluid to be absorbed and usually prevents the rapid accumulation of fluid. The gas is then slowly absorbed and the lung reëxpands.

It has been known for a long time that tuberculosis complicated by sugar in the urine gives a bad prognosis. The X-ray shows conclusively that many such cases have numerous cavities, that they are not limited to the upper lobes, that the cavities have thin walls and that the pus is very apt to burrow in the line of gravity.

Cavities, like pleural effusion, are often easy to diagnose but often physical signs indicate their presence when none exist. This is particularly true with older people. And they often exist without sufficient signs upon which to base a diagnosis. This is more apt to occur in middle age or youth. There is no surer way of diagnosing cavities than by the stereoroentgenogram. By this means we have their size and number, their character and location, and whether or not they contain fluid, all of which is of the utmost importance in diagnosis and treatment.

As yet syphilis of the lung has given us nothing but negative findings. Careful study of our three and four + Wassermann cases has revealed no density upon which it would be safe to diagnose syphilis. The symptoms which have become classical for early pulmonary tuberculosis are rapid pulse, slight afternoon fever, loss of appetite and weight and afternoon fatigue. X-ray studies have emphasized that these should not be classed as tuberculosis symptoms but as symptoms of focal infection. If you add to these cough and expectoration of blood you have a complete picture of tuberculosis, yet these were the symptoms of four hemorrhagic infarct cases all of which made a rapid and complete

recovery. Two had bad teeth, one infected tonsils and one disease of the ethmoid. Yet early tuberculosis is certainly a focal infection of the lungs and these symptoms should always call the chest to our consideration, but without physical signs or a positive X-ray diagnosis the patient should never be condemned to a tuberculous life. Let us rather search elsewhere for the foci.

Almost every one who writes of early tuberculosis refers to the early physical findings, yet it is very unusual for the writer to say definitely what to him are the early physical signs. Correlation of the plate marking with the physical findings has emphasized some findings and has minimized others. Râles at the end of inspiration which do not entirely clear up after cough or which are only heard after exhaled cough and inspiration are of the most value, when restricted to a limited area. Prolonged expiration should never go unnoticed. This will be especially valuable to any clinician who has worked out for himself what to him is the normal proportion between the length of inspiration and expiration in various parts of the chest. If expiration is prolonged and higher pitched than inspiration it adds to the importance of the sign. If expiration is tubular it may not be assured that the lesion is necessarily more severe, but older. Granular or interrupted inspiration may be the only physical sign present. Faint or distant breathing should receive the same consideration. Also increased whispering pectoriloquy over the left upper lobe is a most valuable sign.

I have never known a case to have prolonged expiration with râles limited to the end of inspiration which did not show definite tuberculous densities on the plate near that area. Faint breath sounds may be charged to inability of the normal sound to get through and I have mistaken friction sounds for interrupted breathing but quite commonly they correlate with X-ray findings. The X-ray is most valuable in determining whether these signs are due to change in the lung or pleura. Loss of resonance is a most valuable sign, but does not necessarily mean tuberculosis, as I have known good men to insist. Often we have a loss of resonance and the plate shows a normal density. I have usually considered this due to a thickened pleura which was so evenly distributed as not to show on the plate. Unusual dulness and auscultatory signs of cavity even when they cannot be confirmed by the roentgenogram must be considered. They probably mean a loss of lung tissue or impaired lung function. There is more to be studied in lungs than tuberculosis.

Râles which are heard on inspiration and expiration do not indicate tuberculosis but a catarrhal condition of the air passages. This may or may not be the result of tuberculosis. Such râles will often clear up after rest and I am inclined to believe that when they do oedema should be suspected if nothing abnormal is found on the X-ray plates. La grippe often causes such a condition without plate change but more rarely we have localized pneumonia: then the X-ray plates show a distinct density and usually the physical signs might be described as a wet tubular breathing in contrast to the dry cracklings of pneumonia.

For several years certain cases of la grippe and oedema and a few cases with tubercle bacilli in the sputum gave me no end of trouble to correlate. Clinically and physically they were definitely tuberculous and yet my plates did not show definite densities where the lesion existed. What was the trouble? In the case of oedema I found definite lesions at the apex but none at the base where the moist râles were heard. I concluded that over the area of coarse râles we must have a tuberculous lesion which did not show on the plate. Finally when I observed that these cases promptly cleared up after rest I came to understand that they were not tuberculous. Also with la grippe, with expectoration of bloody sputum and sometimes slight hemorrhages I always diagnosed such cases tuberculous and blamed the plates for not showing it. Later I learned that they never had bacilli in the sputum and that they made an excellent and complete recovery. I now know better the value of my plates.

But cases which expectorated large masses of tubercle bacilli for a short time, and were not very sick but coughed and spat some blood, and showed no fan like markings on the plates, were certainly a great trouble to explain. I watched three such cases most carefully. Two have since expectorated lung stones, all have made perfect recoveries and have been well for four years. Also the plates showed unusual shadows in the hilum or at the tracheal bifurcation. I have concluded that they were cases of mediastinal tuberculosis and that one or more of the nodes had broken into a bronchus and that when the necrotic area had been expelled they recovered. In all cases the cough subsided, and the hemorrhage did not return; one, not until the lung stone had been expelled but the other two after three or four weeks of rest. All have remained well. A fourth has done as well but I have watched him less than six months. I now feel that a favorable prognosis can be made in all such cases when the X-ray plates give a negative finding.

Diaphragmatic pleurisy and pericardial adhesions are frequently noted. The former suggest the possibility of Capps's diaphragmatic pains but pain is not always found. The excursions of the diaphragm, especially in the back, should always be noted. Much loss of motion is usually accompanied by an involvement of the lower lobe or diaphragmatic adhesions. In contrast to this Litten's sign has been of little value. Whether this is the fault of the observer or the sign I have not determined.

As you would expect, by using the stereoroentgenograms it is not difficult to make the differential diagnosis of tuberculosis from most of those diseases which simulate it clinically, but it is difficult to differentiate tuberculosis in this way from any lesion which causes a similar density. Brown induration is such a pathological lesion and is generally caused by mitral stenosis. It is, of course, this difference between the fundamental principles of diagnosis in the physical and clinical examination that make the coördinated method so valuable in analyzing the various points of differential diagnosis in diseases of the thorax.

Prognosis is necessarily more accurately made the more nearly we completely understand the pathological conditions. Then it is necessary to know fully the extent of the lesion, not only how much of the thorax is occupied by infiltrated lung but what percentage of the lung parenchyma has been destroyed.

Surgery of the lung encounters all of the normal difficulties of general surgery, pain, hemorrhage and infection, and in addition, that of air embolism. This latter is a real danger because the pulmonary veins are similar to the arteries in structure and stand open as in no other part of the body. There is a negative pressure from the left auricle which has the potentiality of drawing a continuous stream of air. The phenomenon resulting from air embolism is usually described under the indefinite term of shock. Thus barring extensive pleural adhesions it is easy when advisable to remove an entire lobe, but the removal of a part of a lobe should not be attempted. Further, the drainage of an abscess through the healthy portion of a lung or the aspiration of an abscess through healthy lung tissue should not be attempted, though these operations have both been reported successful.

We have found that in properly selected cases therapeutic pneumothorax is of the greatest value because it puts the lung at rest and facilitates drainage. Also that in certain cases this treatment cannot be applied on account of the pleural adhesions. These roentgen studies

demonstrate what cases necessitate the major operation of resecting the ribs to secure lung collapse.

In pneumothorax and in hydro-pneumothorax sometimes the intrapleural pressure becomes so great as to cause distressing dyspnoea. This can easily be relieved by aspirating the air as well as the fluid and here again we grant the value of the X-ray as guide.

In short all I have said this morning is but the presentation of a few isolated but certainly not unrelated facts that the roentgen examination has not only established but has helped me to interpret. If we had read, marked, learned and inwardly digested Flint's studies we might not have needed our X-ray evidence, but to-day we are more convinced by practical proof than by authority and here indeed seeing is believing.

THE CONE AND COLLATERAL CIRCULATION IN PULMONARY TUBERCULOSIS

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All I wish to say now about the pathology of pulmonary tuberculosis is that the roentgen markings which I have previously described as characteristic of tuberculosis are produced by a cone shaped lesion within the lung, which, like the roentgen shadow, has its base to the pleura and apex to the hilum. This study of the pathology also emphasizes that lymphoid tissue plays the same part in the lung as in the other parts of the body; that is, lymphoid tissue is always so placed as to be a filter through which lymph from the periphery must pass before being admitted to the central ducts and the blood stream. As to the cone, the reason that it has been difficult to find, is because we have been unable to see it inside the lung and whenever we made sections of the cone we have had either a point, a circle, an ellipse or a triangle before us.

To study this pathological lesion we must either employ the X-ray or serial sections. Its cone shape is determined by the anatomy of the lung. The varying densities of this tuberculous cone differ from homogeneous densities of other pathological lesions such as pneumonia, because tuberculosis is a chronic disease and each individually developing tubercle within the cone is limited in size by the nature of its cellular pathology. Thus we frequently find within the cone such varying densities as areas of calcification which are most dense and appear as bright spots upon the roentgen plate; areas of caseation which are quite dense, but not so brilliant on the roentgen plate; and less dense than either of these the mottled effect which is due to cellular infiltration. In contrast to either of these again we have the faint cloud effect suggesting tobacco smoke, which is the result of the acute secondary process, producing tuberculous pneumonia, and in which the air cells are more or less filled with epithelioid and epithelial cells and serous exudate. At another time we find the change in density consisting only of interweaving, which is the result of an old process which has not been able to advance, and in which few of the air cells have been destroyed. When the process is far advanced the area involved increases to such

density that the whole mass appears white, and all lung structure is blotted out. One or all of these processes can exist in the same lung.

Usually pulmonary tuberculosis spreads by numerous subsequent infections which extend from above downward, so we have another distinguishing roentgen characteristic of tuberculosis in the fact that these triangles are of varying densities. This is in marked contrast to pneumonia and hemorrhagic infarct, the shadows of which are homogeneous and the individual cones of which are usually much larger. Acute mucous infections of the lung, such as bronchitis and so called grippe resulting in but slight cellular changes do not produce markings on the roentgen plate that differ from the normal. Malignant tumors differ from tuberculosis because the growth of the tumor is not limited as is that of the tubercle. Thus, especially in sarcoma and frequently in carcinoma, we have large spherical tumors which are in no way limited by the anatomical structure of the lung and therefore their record on the X-ray plate differs evidently in shape from that of the confined tuberculous lesion. The one lesion which is most likely to simulate tuberculosis is the brown induration which occurs in diseases of the heart, such as mitral stenosis.

The characteristic tuberculous cones are quite constant in individuals over twelve years of age, but only occur in children under twelve where the disease is advanced. Usually such cases are accompanied with characteristic physical signs. Many children who have a definite family and personal history of tuberculosis and are evidently suffering from the disease do not show these characteristic markings. Even the more recently perfected X-ray technique has failed to show their presence. This demands explanation. I have said that the cone form was the result of the anatomical limits of the lung. This does not mean only the form limits like those of lobules and lobes. It means also the limits or shape to which anatomy and physiology restrict the spread of the disease. This difference in location and shape of lesion of the same disease in adults and children forces me to believe that primarily tuberculous lesions drain towards the hilum and that the lymphatic system protects the parenchyma. The characteristic adult picture is the result of subsequent infections where the lymphatics have been unable to drain the lesion toward the hilum. In that case the lymph drainage seeks the bronchial nodes by way of the collateral lymph circulation which goes to the pleura and then on to the lymph nodes via the pleural lymphatics. Miller by his anatomical studies, Councilman by his

pathological and Cunningham by his embryological researches have each proved the possibility of this collateral circulation and I want you to see a few of my own sections of tuberculous lungs which show the engorged lymphatics containing epithelioid cells and the valves pointing definitely towards the pleura. And last, I want to call your attention by means of a few slides to the lymphoid tissue and its location near the bifurcation of the bronchus and the vessels, and where it is found in the pleura, and to call your attention too to the fact that the macrophages bring foreign bodies, especially coal pigment, to these areas. These macrophages wander through the lungs generally and may take the infecting organism from the vessels or the air passages. Many of these infected lymphoid centers are near the bifurcation of the bronchus and when broken down have a tendency to break on the surface which, in the lung, is the bronchus. It is my belief that the tubercle bacillus can be one of these foreign bodies and if the macrophage is able to destroy the organism we are safe. If not destroyed, it is deposited in the lymphoid tissue and if it is there destroyed, we are again safe, and even if it continues to multiply, the lymphatics may sufficiently drain this part to prevent the lesion. But if all means fail, a tubercle is formed at this point, and if the infection is sufficiently virulent subsequent tubercles will take place along the line of the lymph drainage. Early, as in children, this drainage is toward the hilum by way of the main trunk. Later, as in adults, it is by way of the collateral circulation towards the pleura and to the hilum nodes through the lymphatics of the pleura.

Thus these anatomical and pathological findings of the cone and the collateral lymphatic circulation account for the roentgen markings that we have declared characteristic of tuberculosis in adults and explain the lack of these markings in children.

PLATE 1

FIG. 1. *1*, Section of cone cut to form triangle; *2*, another cone cut oblong; *3*, area less involved between greater densities; *4*, bronchi leading into their respective areas of different densities; *5*, pleura.

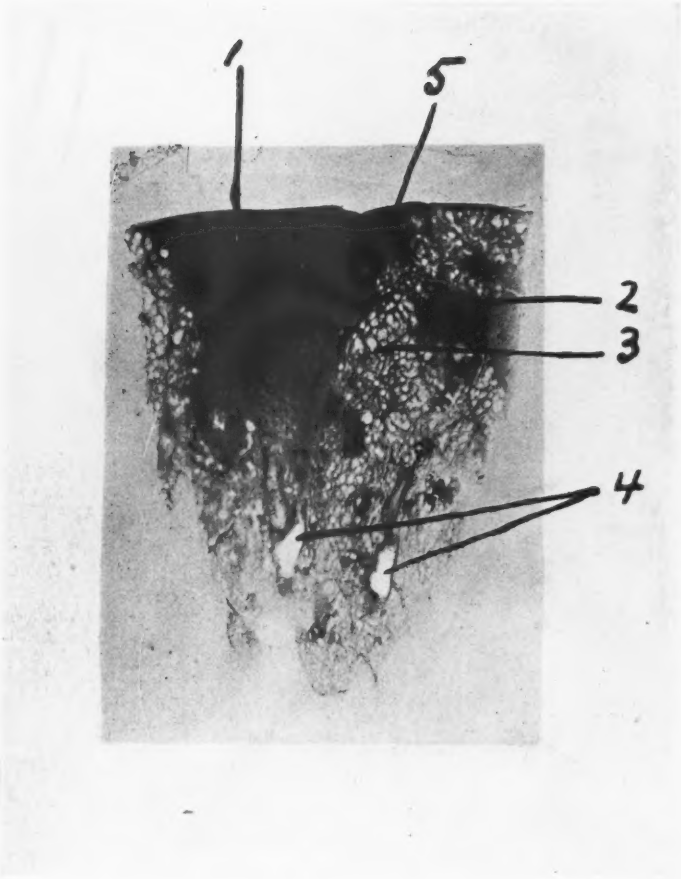

FIG. 1

PLATE 2

FIG. 2. *1*, Pleura; *2*, lymphatics in pleura; *3*, lymphatic in septum; *4*, valves opening towards pleura; *5*, valve in lymphatic of pleura.

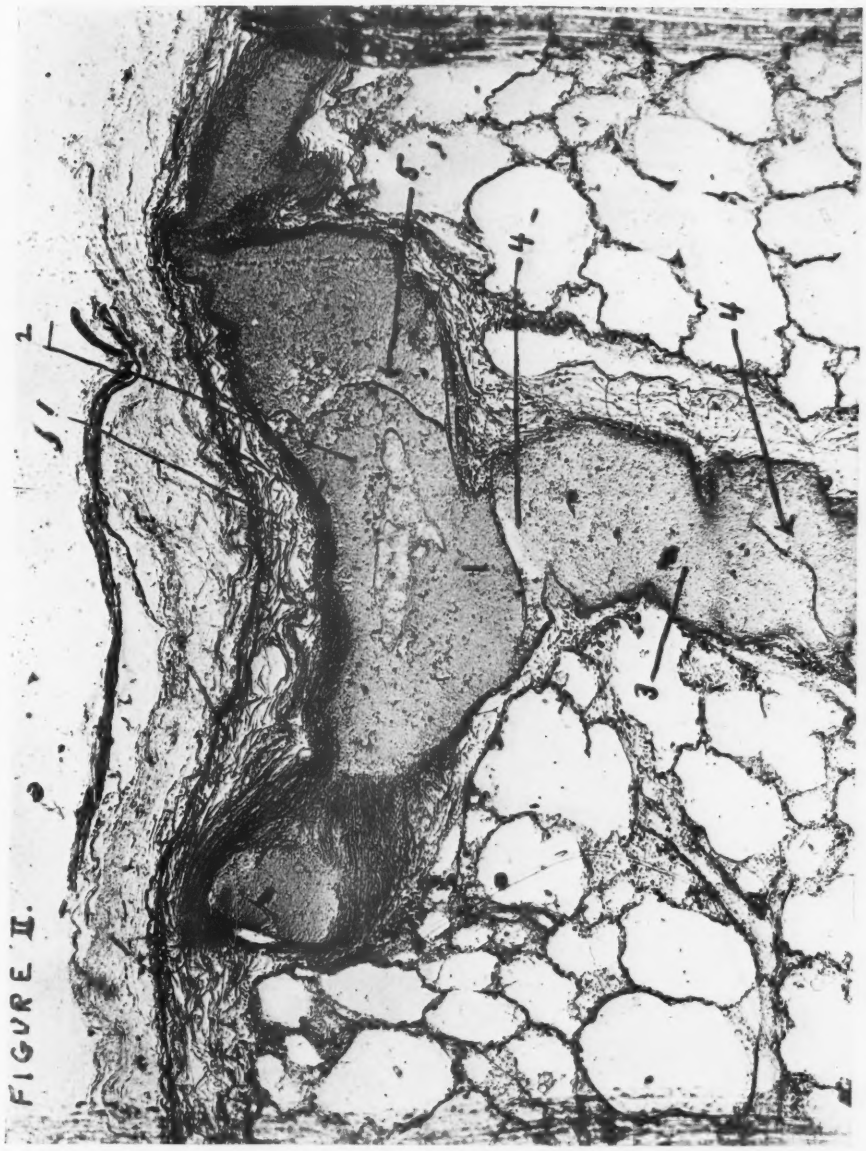

FIG. 2

PLATE 3

FIG. 3. *1*, Pleura; *2*, primary lobule; *3*, trabeculae bounding secondary lobule; *4*, vein with tuberculous mass filling crotch; *5*, bronchus surrounded by tuberculous mass. This block shows no secondary or pneumonic (exudative) process. Thus the air passages are open except where compressed or invaded by cellular growth. This bronchus can be followed to its ultimate air cells, and none of them are filled, infiltrated or obliterated. *6*, large vein in septum; *7*, tuberculous masses. At no place throughout the series do these lesions break through the trabeculae. Often the trabeculae are infiltrated, but they always protect the air-cells on the other side; *8*, lymphatics of the pleura, much distended; *9*, lymphatics in the septa. They can be traced in the serial sections as a continuous vessel from the large vein to the pleura. Epithelioid cells are common in the lymphatic. *10*, area in which valves in septa (fig. 2) are found in another section of this series.

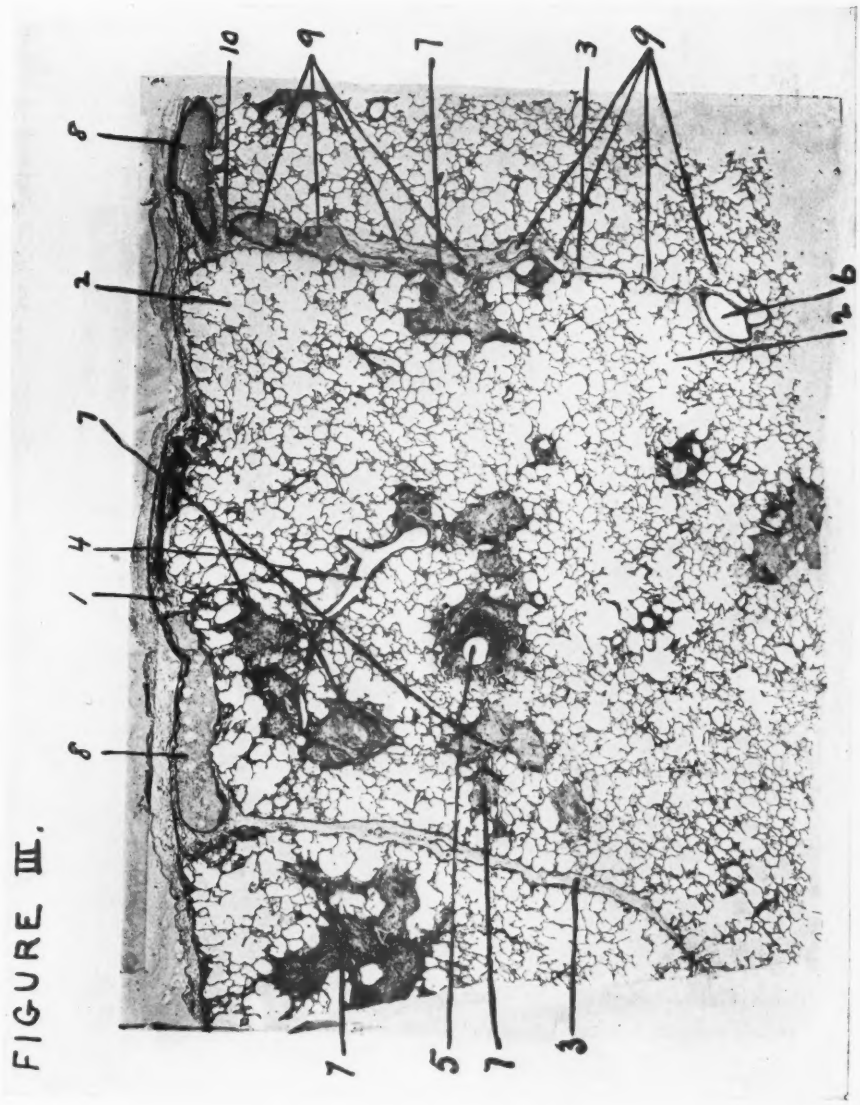

FIGURE III.

FIG. 3

MEDICAL NOTES, ABSTRACTS AND REVIEWS

It will be the eventual purpose of the REVIEW thoroughly to canvass all literature on tuberculosis as it appears in other medical periodicals and to present it in abstract form. Authors are requested to send abstracts or reprints of their papers to the Abstract Editor, Dr. George Mannheimer, 41 West 51st Street, New York, N. Y.

Tuberculosis—First Quarter—1917.—In the following table are presented statistics concerning pulmonary tuberculosis in New York City for the first quarter of 1917, and for purpose of comparisons, figures for the corresponding period of the year 1916 are given. It will be observed that the number of new cases reported was practically the same for both quarters. At the same time there was a slight increase in the death rate from pulmonary tuberculosis for the first quarter of 1917. This is probably associated with the increased prevalence of pneumonia and influenza during the early months of the present year.—*Weekly Bull., Dept. of Health, New York City, Aug. 18, 1917.*

Tuberculosis on Virgin Soil.—Up to 1900, pulmonary tuberculosis was unknown in Central Persia, a plateau varying from 4000 to 6000 feet. About the year 1900 it appeared among the Armenians of Julfa, a village two miles from Ispahan, being introduced into that community by an Armenian merchant who had lived in India. Two years later it appeared among the Mohammedans in the town of Ispahan. Between the years 1900 and 1908 over 100 cases were seen and treated in Ispahan. Two facts stand out with regard to these cases: first, the rapidity of the disease; rarely did it last more than six months; never more than one year; secondly, the mortality;

Statistics for the first quarter of 1917, compared with those for the corresponding period of 1916

		MANHATTAN	THE BRONX	BROOKLYN	QUEENS	RICHMOND	CITY
Population	1916	2,634,223	575,877	1,928,432	366,426	97,883	5,602,841
	1917	2,632,977	599,216	1,975,801	379,696	99,302	5,737,492
New cases	1916	3,186	431	1,363	266	58	5,304
	1917	2,985	472	1,562	232	52	5,303
Total cases in register, March 31	1916	20,429	3,431	8,674	1,557	351	34,442
	1917	19,068	3,239	8,253	1,503	345	32,048
Admitted to hospital	1916						3,693
	1917						3,701
Examined for admission to sanatorium	1916						451
	1917						509
In attendance at day camp	1916	148		93			241
	1917	148		93			241
New patients examined at clinic	1916	1,999	636	1,545	202	26	4,408
	1917	1,825	647	1,536	344	20	4,372
Total visits of patients	1916	14,900	4,649	10,133	2,182	275	32,139
	1917	12,506	4,074	8,562	2,123	262	27,527
Under sanitary supervision	1916	781	179	1,037	24	26	2,047
	1917	667	179	1,024	7	20	1,897
Voluntary renovations	1916	2,189	232	968	77		3,466
	1917	1,831	187	1,058	145	53	3,256
Specimens of sputum examined	1916	10,686	1,897	3,997	669	163	17,412
	1917	9,600	1,908	4,037	718	274	16,537
Of which positive	1916	2,124	294	997	163	36	3,614
	1917	2,156	194	836	108	82	3,376
Deaths, pulmonary tuberculosis	1916	1,195	246	691	140	57	2,329
	1917	1,191	309	812	148	43	2,503
Death rate per 1,000	1916	1.82	1.71	1.44	1.53	2.34	1.67
	1917	1.80	2.09	1.67	1.58	1.75	1.77
Deaths from all tuberculosis	1916	1,380	267	778	157	63	2,645
	1917	1,359	3.44	908	170	48	2,829
Death rate, all forms tuberculosis, per 1,000	1916	2.10	1.86	1.62	1.72	2.58	1.89
	1917	2.06	2.33	1.87	1.82	1.95	2.00

all died. In 1908 treatment with tuberculin was commenced. Up to the end of 1914 close on 500 patients had been treated by this method. The average time occupied by the treatment was six months. The preparations used were tuberculin bouillon filtrate bovine (P. T. O.), old tuberculin bovine (P. T.) and old tuberculin human (T.). The first dose administered was rarely larger than 0.0001 cc., P. T. O., and this was increased until the patient was able to tolerate 1 cc. T. The treatment was then stopped; test injections of 0.5 cc. and 1 cc. T. being given once every three months for the first year and once every six months for the next two years. If at the end of three years no reaction had taken place, the case was considered cured; if reactions occurred, a second and sometimes a third shortened course of treatment was given. Forty per cent were alive and well three years after the last reaction to a large dose of tuberculin. In 20 per cent of the cases the treatment was found unsuitable, and discontinued; the remainder of cases were lost sight of. Previous methods of treatment had proved a complete failure, and in consequence tuberculin was tried in cases which could not be considered favorable, such as mixed infection. Altogether the results are encouraging.—*Tuberculosis on Virgin Soil. H. J. Marrable, Dublin J. of M. Sci., July, 1917, cxliv, no. 347, 25.*

Occupation and Mortality.—The relative mortality from tuberculosis is lower among blacksmiths considered as a group, and also in each age group, than the average relative mortality from the same cause of all occupied persons over fifteen years.

Cigar makers and tobacco workers experience a mortality from pulmonary tuberculosis that is 25 per cent above the average. It is interesting to note that in this group the mortality from tuberculosis is above the average in the groups from fifteen to twenty-four, forty-five to fifty-four, fifty-five to sixty-four, sixty-five and over, and that it is lower in the group between twenty-five and forty-five. It is possible that this difference in mortality at the different age groups has some special significance, but the figures are not sufficiently large to permit of any definite conclusion, and it is most probable that it is but a fluctuation due to paucity of data.

Clerks, bookkeepers, office assistants, etc., have a mortality from tuberculosis of almost twice that of all occupied persons over fifteen years. Examined by age groups, it is found that the mortality from pulmonary tuberculosis is higher than the average at every age group and therefore this cannot be attributed to the preponderance in numbers of persons engaged in these occupations at the earlier ages when the incidence and mortality of tuberculosis are greatest. It is probable that persons who are not robust seek employment as clerks in preference to the more arduous occupations, thus creating an occupation group that is predisposed to disease, but the fact remains that the occupation of clerk, office assistant, and the like is hazardous and that the hazard is pulmonary tuberculosis. The reason for this is not hard to find. Continual confinement in poorly ventilated offices and shops, necessitating the breathing of vitiated air, together with a more or less sedentary life, lowers the physical resistance of these persons and creates a fertile soil for tuberculous infection. There is also a social factor here; that is, the wages of clerks, office assistants, and others of this group are comparatively small and in order to hold their positions they must maintain a good personal appearance; therefore there is a disproportionate outlay for clothing, with the consequent cutting down of the amount of income devoted to food, housing, and other necessities.

The relative mortality for tuberculosis among compositors and printers is still higher than among clerks. In fact, their mortality is more than twice that of all occupied persons of fifteen years and over and is higher in every age group. In this occupation also, pulmonary tuberculosis is unquestionably a hazard.

Garment workers' mortality from pulmonary tuberculosis compares very favorably with that of all occupied persons, 17 per cent of all deaths among this group being the result of this disease. The shop conditions and the wages, the social and economic conditions, of these workers are no more favorable than those of clerks; on the contrary, they are probably less so. Nevertheless, their mortality is but slightly more than the average and practically half of that of the last group mentioned. It would seem that this low mortality may be due to a racial immunity to this disease of the people engaged in this occupation, most of whom are Jews.

The mortality of laborers from pulmonary tuberculosis ranks next to bookkeepers in point of highness. It will be noticed that between the ages of fifteen and twenty-four years the mortality of this group is below the average; at twenty-five the percentage of deaths caused by pulmonary tuberculosis rises above the average; between forty-five and sixty-four the percentage is double the average; and after sixty-five it is more than three times the average. The causes of this high mortality are many. Perhaps the most important is that in this group are to be found

all the misfits who have failed to make good in the other occupations because of drunkenness, carelessness, or ill health, and this also probably explains why the rate is below the average in the first age group of laborers and becomes gradually higher in the later groups.

The mortality of machinists from tuberculosis is higher than the average, but, unlike laborers, the increase grows smaller until it not only disappears but is actually converted into a decrease in the group over sixty-five.

Painters, paperhangers, varnishers, etc., have a relative mortality from tuberculosis that is higher than the average. In the first age group, however, it is lower than the average, but thereafter it rises decidedly above the average.

The mortality from pulmonary tuberculosis among railway track and yard workers is so little above the average as not to require detailed notice—*Occupation and Mortality: their relation, as indicated by the mortality returns in the city of New York for 1914. S. W. Wynne, and W. N. Guilfoy, Publ. Health Reports, June 8, 1917, xxxii, no. 23.*

Miners' Consumption.—A recent investigation of the causes and prevalence of miners' consumption among the metal miners in southwestern Missouri forms the subject of Public Health Bulletin No. 85 issued by the U. S. Public Health Service.

Miners' consumption consists essentially of a mechanical injury to the lungs due to the prolonged inhalation of hard rock dust. It has been recognized as being prevalent in some American mining districts, particularly in the Joplin zinc and lead districts. It was to determine its actual prevalence and its relationship to pulmonary tuberculosis, that the investigation was undertaken.

In the Joplin district certain mines are known as "sheet-ground" mines, in which the ore is found imbedded in an exceedingly hard flint.

In drilling and other mining operations this flint rock is finely pulverized. The minute rock dust particles enter the lungs, in the process of natural breathing, and by their irritating action cause the formation of fibrous, or scar-like tissue. The effect of this is to lessen the lungs' ability to expand and contract, with the result that the victim first notices that he is becoming short winded. With continued exposure to this silica-containing dust, the difficulty of breathing increases, until the miner is no longer able to perform active physical labor. It was found also that men with dust-injured lungs were especially liable to develop tuberculosis, the dust irritation lessening the ordinary resisting powers of the lungs. While miners' consumption is not in itself infectious or contagious, it predisposes to tuberculosis. The greater the amount of rock dust injury the greater the liability to tuberculosis; the far-advanced cases of miners' consumption practically all become tuberculous before their death.

Under an entirely voluntary system 720 miners presented themselves for physical examination, of whom 433 were found to have had their lungs injured by the inhalation of rock dust; of these 103 were also tuberculous, the amount of tuberculosis infection being greatest among the advanced cases of the rock dust disease.

Five years' steady work with exposure to flint dust is fairly certain to find the miner in at least the first stages of miners' consumption. If the miner continues his work after being affected, death usually results within ten years from the time that exposure to flint dust commenced. Poor housing conditions were found to be prevalent and to add to the liability of tuberculous infection. Apparently tuberculosis is now occurring at an earlier stage of miners' consumption than was formerly the case. The report lays emphasis on the necessity of preventing the spread of tuberculosis through these cases, especially among miners' children. The fact that miners' consumption is a forerunner of tuberculosis necessitates that it be treated with the same hygienic precautions as is the latter disease.

The report concludes that aside from the hygienic supervision of underground working places, the education of the miner against the spread of infection and supervision of miners' children, especially those of consumptive parents, are matters of vital importance.—*Editorial, Boston M. & S. J., June 28, 1917, clxxvi, no. 26, 904.*

Nomenclature.—In the nomenclature of tuberculosis we should keep clinical conceptions and pathological conceptions out of the same pew, and then employ appropriate and unmistakable terms to express those ideas. The term "incipient" when used in the pathological sense, as "incipient tuberculosis" denotes the primary infection of which we are in most cases ignorant, the patient knowing nothing about it; "incipient phthisis" means the manifestation of the actual disease. Early clinical tuberculosis of the lung cannot be designated as "incipient" because it is a metastatic process which has begun years before. The term "active" when not otherwise defined should be used to denote a clinically active case, subjective as well as objective. The term "quiescent" denotes absence of activity and not slightly

lessened activity. To denote the latter, the term "less active" may be employed. "Latent" is the condition before it has become active; after an active case has become inactive we speak of it as "quiescent." We still need a term to take the place of "tuberculous patient."—*The Nomenclature of Tuberculosis. W. W. Dill, Southwest. Med., March, 1917, i, no. 3, 21.*

Fear of Tuberculosis.—Prior to the establishment of the infectiousness of tuberculosis, when the disease was considered hopeless, phthisiophobia was not nearly as widespread as with the new era of research and study. The interest displayed by the public and by the profession in the study of tuberculosis, brought with it an unwarranted fear of the disease. As a result he who is tuberculous carries with him a stigma, is shunned by all, and barred by society and business.

The possibility of hereditary transmission of tuberculosis is practically nil and can safely be ignored. A poor physique in an environment favorable for the development of tuberculosis is of far greater importance. The disease is not contagious in the sense of smallpox, it is communicable, not by a chance contact, but by repeated or prolonged exposure. Then again the tuberculous infection suffered in early life actually has a relative minimizing effect in later life The disease certainly is on a decline since 1885. It is waning both in frequency and mortality. Better means of early diagnosis and treatment, and almost universal study and investigation of this disease, account for this change.

In the face of all this, phthisiophobia surely is unwarranted and should be overcome by all means. The solution of the problem is in the hands of the general practitioner. He comes in daily contact with the victim of this illogical dread. He should enlighten the public on the nature of tuberculosis. He should tell them that though childhood infection is the rule, it by no means results invariably in subsequent clinical tuberculosis. He should preach that the campaign for better health in general is the best means of preventing tuberculosis. Periodical examinations should be instituted, both of children and adults, and the necessary instructions given when indicated. The once over-accentuated danger of universal infection was responsible for the prevalence of phthisiophobia, and it is up to the general medical man to overcome it.—*The Unwarranted Fear of Tuberculosis. C. H. Cocke, South. M. J., July 1917, x, no. 7, 5395.*

Tuberculosis Plus.—Tuberculosis is seldom if ever a pure and isolated infection. As such it would destroy very few lives and would require a long time to do it. A cold abscess does little harm and is slow in doing it. Tuberculous glands may exist for a long time and give practically no trouble. The same may be said of tuberculosis of the lungs, the spine, skin, bone and peritoneum. Add pus-producing organisms to pure tuberculosis and trouble begins. Glands break down, lung tissue is replaced by cavities, ostitis manifests degeneration, and Koch's bacillus can be demonstrated in the discharges from tuberculous epididymis, testicle and kidney. This is tuberculosis plus. Tuberculosis works very slowly; tuberculosis plus gets in its work very rapidly. Treating tuberculosis is not enough. The plus must have scientific attention. The measures which affect tuberculosis favorably have no influence on the plus. It is very important that we do not produce a plus where simple tuberculosis exists. Such secondary infections are often produced by surgical intervention especially after ether anesthesia. When surgical intervention is absolutely necessary a local anesthetic should be employed; if this is not feasible, gas is the only logical anesthetic. Other pluses besides infection with pus producing organisms are: gastrointestinal disturbances, loss of appetite, and other diseases. Forced feeding is absolutely wrong, since the extra food will not be digested. For this reason bitter tonics are useless. Rest in bed will improve the appetite. Homesickness and other mental worries are also serious pluses. For this reason it is unwise to send patients away from their relatives, especially without adequate financial means.—*Tuberculosis Plus. A. Henry, J. Ind. State Med. Ass., February 15, 1917, x, no. 2, 59.*

Interest in Tuberculosis by the Medical Profession. Much is yet to be learned about tuberculosis. The time will surely come when tuberculosis will be conquered. Possibly the solution will be found in periodic physical examination of all persons, and the isolation of children from tuberculous adults. The general practitioner can do most along this line. However, the average doctor fails to bestow upon this subject the intelligent study that it deserves. He fails to utilize the means at his disposal to make an intelligent diagnosis. Less than 2 per cent of the admissions to the Fort Stanton Sanatorium had been correctly diagnosed at the different government transferring stations by the highly trained officers stationed there. What can, there-

fore, be expected from the ordinary practitioner?

The early case: Incipient pulmonary tuberculosis consists of an infiltration of one or both apices or of a small portion of one lobe, without destruction of tissue, marked constitutional symptoms or serious complications. Fine râles are usually detected, and possibly some change in palpatation and percussion. Sanatorium treatment in this type is not necessary. Rest, preferably in the open air, good food, and absence of worry, will accomplish the result at home.

The moderately advanced case: Consists of an infiltration or partial consolidation of not more than two lobes, without cavitation or serious complication. There is usually cough and expectoration, moderate pyrexia, malaise, failing appetite, and moderate loss of weight. There is increased fremitus and impairment of resonance, weakened breath sounds or possibly broncho-vesicular breathing. Râles are always present. This type does best in a sanatorium. In patients above forty years with a long standing disease and only slight activity, a modification of the habits of life is all that is necessary.

The far advanced case: In this type more than two lobes are diseased or one or two lobes with cavitation or a serious complication. The great majority of these cases are sufficiently ill and look tuberculous. The physical signs depend upon the underlying pathologic changes. Not much can be done here. Arrest the progress of the disease, if that be possible, prolong his life, and make him as comfortable as possible and keep him from being a menace to others. Sanatorium or hospital treatment will accomplish this, and the nearer the sanatorium the better.—*A Plea for a More Intelligent Interest in Tuberculosis on the Part of the Medical Profession. F. H. McKeon, Southwest. Med., April, 1917, i, no. 4.*

Coöperation from Dentists.—One of the difficulties met with in connection with the proper care of tuberculous persons is that of securing dental service. Proper care of the teeth, important for all persons, is especially required for such patients. Patients whose sputum contains tubercle bacilli are not, and should not be, received and treated in a public free dental clinic.

It is a pleasure, therefore, to record that, through the Consumptive Jewish Aid Society in the Borough of Brooklyn, one of the Department's most active aids in the work of the Tuberculosis Day Camp "Rutherford," arrangements have been made by which a dentist of that Borough will give his services for such dental work as may be required for the children patients of the Camp.—*Weekly Bull. City Dept. of Health, April 28, 1917.*

Tuberculosis Following Ritual Circumcision.—Report of a case of tuberculous infection of the operative wound following ritual circumcision. The infant had been circumcised on the eighth day, and came under observation at nine weeks. There was noted enlargement of the inguinal glands and in the operation scar a few small shot like lesions which microscopically showed numerous tubercles. The operator had advanced pulmonary tuberculosis.

This case makes a total of 42 reported cases. The author reviews briefly the literature and sums up as follows:

From seven to fourteen days after circumcision, the wound which in the majority of cases does not heal, ulcerates and begins to discharge pus; from two and one-half to eight weeks after the circumcision the inguinal glands become enlarged and in the majority of cases break down. Most of these infants die at from three and one-half months to three years after the infection; the most common causes of death are tuberculous meningitis or general miliary tuberculosis; those who recover invariably present other manifestations of tuberculosis in later life. The treatment of these cases is early excision of the tuberculous tissue of the penis and the inguinal glands.—*Mark S. Reuben, Arch. of Ped., 1917, xxxiv, 186.*

Dogs as Tuberculosis Carriers.—The dog is the most persistent, the most ubiquitous, and the best qualified carrier of tuberculosis. He gnaws every decaying bone and feeds on all rotten carcass. He licks and touches everything filthy, following which he licks his master's hands and the faces of children. He furnishes the moisture and warmth favorable for the tubercle bacilli. Every dog is a living menace of tuberculous infection.—*Dogs as Carriers of Tuberculosis. M. W. Vandenburg, M. Times, Aug. 1917, xlv, no. 8, 230.*

Bovine Infection.—Up to 1914, there had been reported 2051 cases of human tuberculosis in which the type of the infecting bacillus had been determined in reliable fashion. One thousand eight hundred and forty-eight cultures belonged to the human, 189 to the bovine type, and 14 to both types together. Aside from 12 cases of skin tuberculosis in butchers, the large majority of bovine infection was of the alimentary variety and comprised 114 cases of tuberculosis of the abdominal

organs and of the cervical and axillary lymph nodes. One hundred and fifty-one out of the 189 bovine cases were children under sixteen years of age. Figuring on the relative frequency of tuberculosis of the lungs and that of other organs, the percentage of bovine infection would amount to about 1.8 per cent. Why English authors find so many more cases of bovine infection than German and Norwegian investigators, is still a matter of special study. The teaching of Robert Koch, that in the fight against tuberculosis the main reliance has to be placed in the prevention of transmission from man to man, still holds good. But the extensive investigations carried on all over the world, have proved that bovine tuberculosis is not a negligible quantity, especially for children. The sanitary supervision of cattle and the procuring of clean milk are important aims of the propaganda. —*Der Typus der Tuberkelbazillen bei menschlicher Tuberculose. Moellers, D. m. W. 1916, No. 33 (translated from Internat. Centralbl. f. d. ges. Tuberk.-Forsch., xi, no. 1, January, 1917).*

The Tuberculous Milch-Cow.—Reviews briefly the incidence of bovine bacilli in human infections and points out the inadequacy of Massachusetts laws for controlling bovine tuberculosis.—*A Common or Public Nuisance—The Tubercular Milch Cow. J. F. Winchester, Am. J. Pub. Health, January, 1917, vii, no. 1, 83.*

Apical Tuberculosis of Adults and Focal Tuberculosis of Children.—Fatal pulmonary tuberculosis after the tenth year originates in the apices. Focal infections of adults occur probably in almost 100 per cent, and usually originate in childhood, in more than 50 per cent between ten and eighteen years. Between eighteen and thirty years at least 85 per cent have acquired focal tuberculosis.

Apical lesions appear in later childhood and occur with increasing frequency from adolescence to old age. Encapsulated and healed tuberculosis increases with age, whereas there is no similar increase in active tuberculosis. After the tenth year focal lesions are almost invariably encapsulated or healed.

The apical lesion in those who have previously acquired focal tuberculosis pursues a chronic course, and in most cases remains latent or is completely healed. Apical tuberculosis without preëxisting tuberculosis may be accompanied by tuberculosis of the regional lymph nodes, whereas apical tuberculosis with preëxisting focal lesions is not followed by tuberculosis of the adjacent nodes.

In apical tuberculosis the lesion is in contact with the pleural surface, the latter being puckered up and frequently drawn into the lesion. It is not likely that tuberculous infection is transmitted by the lymphatics from the focal lesion of the lung or regional nodes to the apex.

The following observations indicate that apical tuberculosis of adults is not the result of infantile tuberculosis, but caused by subsequent infection: (1) Apical tuberculosis does not have its highest incidence in early adult life, but is more common in later life, when focal lesions of childhood have in most instances completely healed. (2) Most of these apical lesions pursue a chronic course. (3) Childhood tuberculous lesions found in adults with apical lesions are usually calcified and healed, whereas the apical lesion is fresh and caseous. (4) In a large proportion of cases of associated focal and apical tuberculosis the focal lesion is in one lung and the apical in the opposite apex.—*The Relation of Apical Tuberculosis of Adults to the Focal Tuberculosis of Children. E. L. Opie, The J. of Exper. M., Aug. 1, 1917, xxv, no. 2, 263.*

Primary Lesion, Secondary and Tertiary Lung-Tuberculosis. 1. Primary Lung-tuberculosis in Man.—The primary lesion in the lung is a small, round, frequently calcified focus (conglomerate tubercle) which is never connected with the pleura but with the regionary lymphnodes which are also diseased and often partly calcified. The further away from the primary focus, the less are the lymphnodes affected. Ranke found such primary lesions (1) at the autopsy of three children, who had succumbed to other diseases; (2) in a fourth child, in whom no focus in the lung, but only in the lymphnodes could be detected; (3) in a child, in whom the portal of entry was ascertained to be in the mucous membrane above the bifurcation of the trachaea; (4) in a seventy-two-year-old man. From the latter case it appeared that anthracotic tissue can offer a certain amount of resistance to the progress of caseation, but cannot hold it back. For obvious reasons, such primary foci occur much more rarely at an advanced age than in childhood. Macroscopically, they appear as calcified cheesy areas surrounded by a dense connective tissue capsule. Microscopically, one sees alterative, exudative and proliferative processes. The former are due to the action of toxins, the latter mainly to that of foreign bodies.—*Primaraffekt, sekundaere und terti-*

aere Stadien der Lungentuberkulose. 1. Die primaere Lungentuberkulose des Menschen. Karl Ernst Ranke, D. Arch. f. Klin. M. 119, 1916, H. 3, S. 201. (Transl. from Internat. Centralbl. f. d. ges. Tuberk. Forsch., Dez. 12, 1916, x, no. 11, 322.)

Histological Examination of Hilus Lymphnodes. II. Generalized Tuberculosis and Localized Phthisis—Ranke made extensive histological examinations on a large series of cases of generalized tuberculosis and localized phthisis. The characteristic criterion of the former is the formation of hematogenous metastases. The changes in the lymph-nodes can be followed up in three stages. In the primary the disease remains localized in the interior of the lymphnodes. In the secondary stage it goes beyond the limits of the node and spreads in the vicinity in the form of an intensive inflammation and rapid caseation of the fresh foci. These changes are found in the compact conglomerates of the primary period as well as in those lymphnodes which drain the hematogenous metastases. In the tertiary period the regional nodes are not much affected, torpid foci forming with little or no perifocal inflammation. On histological examination the primary nodes show remnants of glandular substance embedded in cheesy material. The secondary nodes are completely caseated or they consist of a mixture of cheese and remnants of connective tissue fibers (so-called, "potato-glands"). The secondary stage is characterized by heightened susceptibility to toxins and therefore called by the author the anaphylactic period. In contrast thereto, the tertiary period and the localized forms of phthisis are expressions of a pronounced immunity. Localized phthisis is by no means a continuation of the tertiary stage of generalized tuberculosis; it rather skips the secondary and tertiary stage and arises from the primary lesion after a long interval of latency. Evidently these infections are mild. They spread by way of the bronchial tree. This remarkable fact is explained by the humoral immunity which characterizes this type of lesion. Bacilli which find their way into the blood- and lymph-channels are almost all destroyed there. A few weakened ones may penetrate into the lymphnodes and produce mild non-progressive lesions. These are a few submiliary nodules without perifocal inflammation, without formation of connective tissue, without calcification and without tendency to spread. It is impossible to distinguish recent and old foci. In the lung itself chronic localized lesions are found. By endobronchial propagation these foci become confluent, grow, break down and lead by means of the sputum to an infection of the trachea, larynx, pharynx, nose, mouth and of the gastro-intestinal tract by means of swallowed sputum. The blood-vessels remain pervious for a long time and obliterate only when the external layers of their walls become cheesy. This implies the danger of rupture and consecutive hemorrhage. The entire picture demonstrates that there must be predisposing influences which give rise to a general weakening of the system. On account of its pronounced chronic course localized phthisis offers the best chances for treatment, especially surgical intervention.—*Primaeraffekt, sekundaere und tertiaere Stadien der Lungentuberkulose auf Grund von histologischen Untersuchungen der Lymphdruesen der Lungenpforte. II. Lungenerkrankungen bei generalisierter Tuberkulose und isolierter Phthise. Karl Ernst Ranke, D. Arch. f. klin. M, 119, 1916, H. 4/6, S. 297. (Transl. from Internat. Centralbl. f. d. ges. Tuberk. Forsch., Feb., 1917, xl, no. 2, 35.)*

Transmission of Tuberculosis.—Two types of tubercle bacilli cause disease in man, the human and bovine. In New York City almost all adult tuberculosis is due to the human type. In infancy 10 per cent are caused by the bovine type, and in children 60 per cent of glandular infections. Droplet infection is more important than dried sputum. The dust of rooms inhabited by tuberculous persons is slightly or moderately infected with tubercle bacilli. Whenever sputum clings on lips, fingers, or clothing of the patient, there is danger of bacilli being conveyed to another person; when contact is close the danger is great; when limited, as shaking hands, etc., it is slight. Water infection is probably not great.

The bacilli, whatever their source, may all lodge in the tonsillar areas. There the channels separate. Some bacilli are swallowed, pass to the stomach and may be absorbed by the mucous membrane of the intestines. Lesions may develop at the point of entrance or in the first lymph glands. Inhaled bacilli may lodge in the pharynx or are implanted lower down in the respiratory tract. They cause a local lung infection or are transmitted to the bronchial lymph nodes. At any time the primary infection may spread by the blood and cause secondary foci. Later pulmonary disease is due to dissemination from the primary focus. In most people a focus of infection is developed and this focus gives considerable immunity making a secondary infection difficult.—*Channels of Infection and Methods of Transmission of Tuberculosis. W. H. Park, N. York M. J., April 28, 1917, cv, no. 17, 777.*

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* "The Choice of Tuberculins," by Benjamin White, Otisville, N. Y., Department of Health, City of New York. Read at the annual meeting of the Medical Society of the State of New York, at Saratoga Springs, May 18, 1916.

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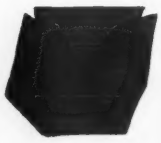

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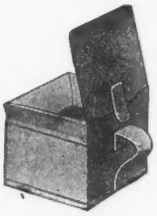

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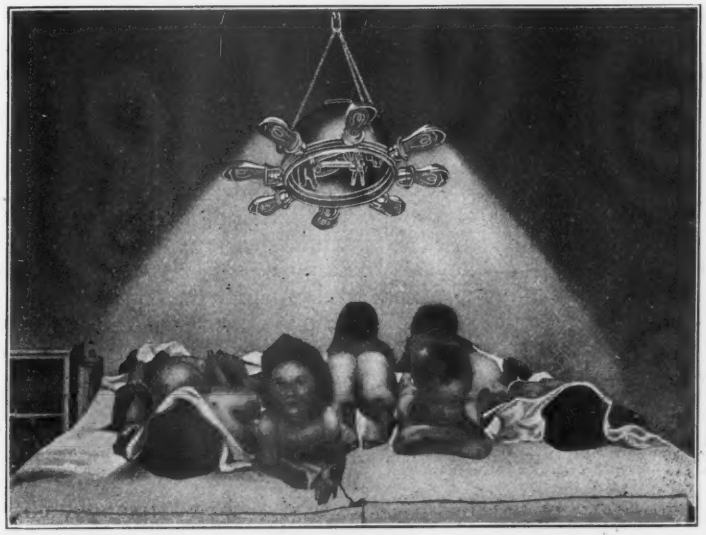

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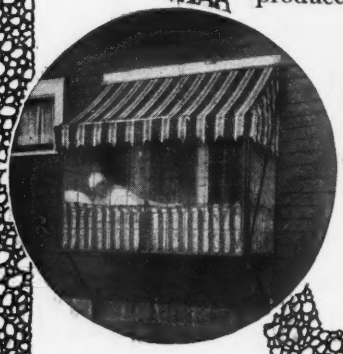

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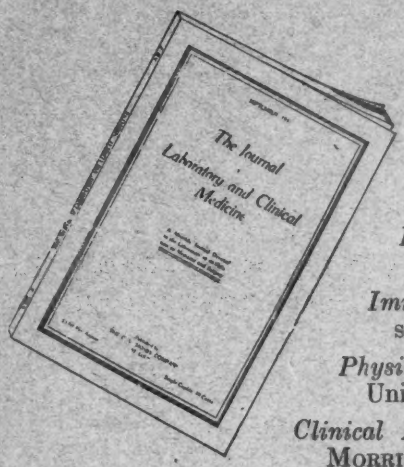

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